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DISTORTIONS OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOUR.

(Fourth Paper.)

CHIASMAL LESIONS, WITH ESPECIAL REFERENCE TO BITEMPORAL HEMIANOPSIA.

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Introduction: Source of Material.

The preceding papers in this series were devoted (1) to a statistical review of the perimetric studies in a series of 200 cases of brain tumour observed at the Johns Hopkins Hospital between June 18, 1905, and January 1, 1911 [7]; (2) to the deviations, particularly of the colour fields, in relation to the advancing stages of choked disc in the same series of cases[1] [8]; and (3) to the question of binasal hemianopsia and the tendency of this deviation to characterize the last stages of a declining choked disc [9].

In the first paper of the series it was stated that the use of the perimeter was possible in 123 of the 200 consecutive cases on which our statistical studies were based, and that in six of these cases a more or less typical bitemporal defect was found. Thus in 3 per cent. of the entire early series, or 4·9 per cent. of the cases in which perimetry could be used, the distortion of the fields indicated a median involvement of the chiasm. These figures, therefore, represent the average occurrence of bitemporal hemianopsia in the general run of brain tumour cases, for up to January, 1911, the number of hypophysial growths in the tumour series was not disproportionate, as it has since become. Thus in the 130 additional patients with brain tumour

[1] This question of colour interlacing, upon which we place less importance than formerly, will be further reported upon by one of the present authors (Walker).

observed in Baltimore between January 1, 1911, and May 1, 1912, the number of pituitary cases so far increased as to greatly alter what might be considered the average percentage figures; and this disproportion has been even more marked in the Boston series of 134 cases at the present writing, July 1, 1914.

In the analysis of our "brain tumour" cases, we were from the first led to include a number of patients with hypophysial or infundibular growths—growths which, though intracranial in a strict sense, represent purely extracerebral lesions, distorting the brain proper either not at all or very late in their progress. The number thus included, however, does not represent all the examples of hypophysial disease in the series, for only 101 of the 148 cases observed up to July 1, 1914, deserve, on the basis of neighbourhood pressure symptoms, to be classified among the intracranial tumours, and in only 81 of these were the fields of vision affected. The remaining cases comprise acromegalics and others who have thus far at least failed to show clinical signs of involvement of the chiasm.[1]

From the foregoing it can be seen, in the first place, that perimetric distortions are wanting in nearly half of the examples of hypophysial disease in the stage at which they are commonly recognized to-day;[2] and in the second place, that in about 20 per cent. of the patients actually showing characteristic neighbourhood symptoms there occurs no perimetric change demonstrable by our present methods. It may be gathered, therefore, that the chiasmal "cross-roads" are capable of considerable distortion by a growth in the interpeduncular space without a demonstrable encroachment upon the fields of vision. This, moreover,

[1] In every patient suffering from a pituitary disorder, as has been elsewhere emphasized by one of us ("The Clinical Aspects of Dyspituitarism," The Harvey Lecture, December 10, 1910), there are three separate clinical factors to be distinguished: (1) The *neighbourhood signs and symptoms*, shown by evidences of pressure on adjoining structures—chiasm, oculomotor and olfactory nerves, uncinate gyri, crura cerebri, and the like—and often by a demonstrably enlarged sella turcica; (2) the *general pressure* symptoms in case the growth actually becomes large enough to cause considerable cerebral deformation or to obstruct the cerebrospinal fluid circulation by blocking the foramina of Munro; and (3) the *constitutional* or *glandular* symptoms, either on the side of over- or under-hypophysial activity.

The first and second of these factors alone are of interest in connection with perimetry, for one of the most striking of the neighbourhood signs—indicating chiasmal pressure—is represented by a supposedly characteristic distortion of the visual fields associated with so-called primary optic atrophy. In some cases, however, a choked disc may become superimposed on the primary nerve lesion, with changes due to a so-called secondary atrophy, which may modify the perimetric outlines according to the principles set forth in the earlier papers.

[2] Doubtless these figures will fall markedly as more precocious diagnoses are made, based on constitutional disturbances rather than on neighbourhood symptoms.

has been demonstrated in a few cases in which supposedly normal fields were plotted shortly before death, and yet in which an extraordinary degree of deformation and elongation of the chiasm was disclosed *post mortem*. And what is of still greater moment, a *bitemporal* hemianopsia, or a tendency in this direction, is by no means the necessary expression of the chiasmal involvement when vision does become affected by a hypophysial lesion, for nearly half as many cases in the series have shown an equally definite *homonymous* hemianopsia or a tendency in that direction. In its so-called typical form furthermore, with a practically vertical meridian which sharply divides in each eye the blind nasal from the seeing temporal retina, a *bitemporal* hemianopsia is far from the most common form of field defect. Hence the conclusion that an existent clean-cut bitemporal hemianopsia is an evidence of a medial lesion, most often due to a tumefaction of the hypophysis or its stalk, is justifiable enough; but the reverse argument does not hold—that the absence of this characteristic defect, or indeed of any defect whatsoever in the fields, necessarily speaks against a primary pituitary or interpeduncular growth.

Though it will be fully emphasized that the important feature of these field deformations due to chiasmal lesions is their progressive nature, nevertheless it may be well to give some figures in regard to their relative frequence, and possibly the best fixed point is the time of the patient's hospital admission, representing at the present day, unhappily, a fairly well advanced stage. For our present purposes these patients may be divided into four main groups: (1) those with a bi-temporal defect of vision; (2) those with a homonymous defect; (3) those already blind in one or both eyes, under which circumstances it is often difficult and sometimes impossible to tell in which group—bitemporal or homonymous—the case actually belongs; and (4) the cases with irregular defects, which are scarcely to be classified. In these four groups the 81 examples in our series fall as follows:—

(1) *The bitemporal group.*—Here are included 26 patients, of whom 7 showed acromegalic changes, the other 19 being examples of primary hypophysial insufficiency. In four patients a characteristic bilateral temporal hemianopsia was present with fairly vertical meridian, though in only one instance was the degree of macular involvement practically equal in the two eyes. In nine cases there were fairly symmetrical defects in the temporal fields, but the stage of a vertical meridian was not yet reached or had been passed by. In another nine patients a tendency towards a bitemporal field constriction was evident, but the field defects

were very asymmetrical on the two sides. It may be added that in several cases (necessarily excluded from these statistics) the fields on the first admission were normal but on subsequent examinations the early grades of an advancing temporal constriction began to appear.

(2) *The homonymous group.*—Of the twelve cases included here, four being acromegalics, the defect in three was typical, with a vertical meridian separating the blind from the seeing retinæ. In four cases there was a symmetrical defect in a stage short of the vertical hemianopsia, and in the remaining four an unmistakeable homonymous defect was evident, though the process was very unequal in the two eyes. In two of them normal vision in one eye was associated with a nasal defect in the other—an early stage of what ultimately became a homonymous defect.

(3) *The amaurotic group.*—This group comprises 35 patients, only three of them acromegalics, who on admission were blind in one or both eyes. (*a*) There were four who according to the history, or from *post-mortem* findings, were shown to belong in the group of bitemporal cases. In one instance there was a homonymous return of vision after operation, and it is uncertain in which group the remaining six belong. (*b*) There were 24 patients totally blind in one eye and showing on the other side either a practically normal field (three cases), or a typical temporal hemianopsia (seven cases), a tendency towards a nasal defect (one case), or an atypical field distortion, making it difficult to tell whether the case belonged in the homonymous or bitemporal group (three cases). The same uncertainty on admission pertained to the 17 cases showing a temporal defect which might have been associated originally either with a primary nasal or a temporal blindness in the other eye, but in nine cases, either by the history or by post-operative return of vision, the condition was shown to have been bitemporal.

(4) *The unclassified group.*—Here are included eight cases in which bizarre fields accompanied known pituitary lesions. They comprise cases with central scotomas associated with normal or more or less normal peripheries, one case with a fairly sharp superior hemianopsia, and others with more or less irregular outlines of indeterminate nature, some of them representing our early experiences with perimetry which leave some doubt as to the exactness of the plotted records.

These figures, be it recalled, refer merely to the findings on admission, and before we became familiar with the early gradations of the process in but few of the cases would we have ventured, on the basis of the perimetric studies alone, to predict the presence of a retrochiasmal

lesion. As will be pointed out, the characteristic features of the chiasmal implication in these patients is the progressive involvement of the fields for colour as well as those for form and the associated alterations in central vision (acuity and scotomata). We hope to show what are the earlier perimetric indications of the chiasmal lesion, for the tyro knows the significance of a bitemporal hemianopsia at the stage when a vertical meridian divides blind from seeing field.

Though it was our original intent to include in this report all the cases showing field defects with primary atrophy due to a direct involvement of the chiasm by a hypophysial, or interpeduncular tumour, we have thought it best to reserve the cases with homonymous hemianopsia due to pressure of a tumour against the side of the chiasm for a separate report, since, owing to their presumed rarity—only a few examples are on record—they deserve special emphasis. The present paper, therefore, concerns itself with the cases exhibiting obvious bitemporal defects, together with those in the amaurotic group, which in all likelihood belong also in this category.

Bitemporal Hemianopsias in general.

Incidence.—Though first noted by Mackenzie in 1835, and known to von Graefe in 1856, surprisingly few cases of bitemporal hemianopsia were recorded until the present interest in hypophysial disorders was awakened. In 1880 43 examples were gathered by Wilbrand, some of doubtful chiasmal origin. Griffith, six years later, added 15 cases, four in his own experience; and Seli in 1894 brought the recorded number up to 70, but even at that time very little emphasis was laid on hypophysial lesions as a causal agency. In an elaborate statistical study in 1912 [5] Bogatsch added 34 cases from Uhthoff's clinic in Breslau, and stated that he had record of 315 cases in the literature. In illustration of the growth of our knowledge of pituitary disorders it may be added that according to Bogatsch, of the 59 cases reported before 1886, the date of Marie's description of acromegaly, only 5 per cent. were accredited to hypophysial lesions, whereas in 40 per cent. the cause was said to be unknown, and that in the 256 cases reported subsequently 50 per cent. were ascribed to the hypophysis and only 13 were of unknown etiology. With but few exceptions, in our series the hypophysis or a lesion in its neighbourhood associated with or affecting the hypophysis, such as the interpeduncular tumours which arise on a congenital (pituitary) basis, has been the causal factor.

In the experience of various writers bitemporal hemianopsia occurs in the proportion of 1 to 25 in all cases of hemianopsia, including, of course, the frequent examples due to cerebral vascular lesions. In our statistics, confined to the hemianopsias associated with intracranial tumours, there were 80 examples (including 17 so-called binasal hemianopsias) in the 330 cases observed between June 18, 1905, and May 1, 1912. In these 80 examples there occurred 23 of bitemporal involvement—a proportion of about 1 to 3. This high percentage of bitemporal hemianopsias—which would be even greater did the computation include the Boston cases—is attributable not only to the fact that the series comprises an unusual number of hypophysial cases, and includes moreover, as "bitemporal," cases showing a tendency only toward this defect, but also to the fact that the cerebral vascular lesions, doubtless the most common source of homonymous hemianopsia, naturally are not represented in the series except as an occasional case with a pseudo-tumour syndrome.

Mode of production.—Despite some difference of opinion in regard to the results of various experimental observations on the degree of completeness of the chiasmal crossing in higher animals,[1] the painstaking observations of Henschen [15] on carefully selected clinical material furnish the basis for the most generally accepted ideas in regard to the disposition of fibres in tract, chiasm and optic nerves. Though Henschen's figures, modified slightly by Wilbrand and Saenger, are familiar enough to ophthalmologist and neurologist, nevertheless for the purpose of reference we have ventured to insert new diagrams in this context (fig. 1) as an aid in the interpretation of what is to follow.

As de Schweinitz and Carpenter have pointed out [11], there are probably many individual human variations, all the way from an entire absence of crossing (of which we have seen one example in association with a congenital suprasellar cyst) to a possible total decussation, representing a reversion to the lower and more primitive type of chiasma. In the macular bundle, in particular, variations may occur, some even believing that it has a bilateral cortical representation, and as the question of inclusion or sparing of the macula has been thought to be of considerable importance from a localizing standpoint it is well to have the anatomical relations of the various bundles clearly in mind.

There are three pairs of fibre-bundles or fasciculi to be considered, the uncrossed, the crossed, and the central or macular bundles.

[1] The question is thoroughly reviewed by L. F. Barker [1].

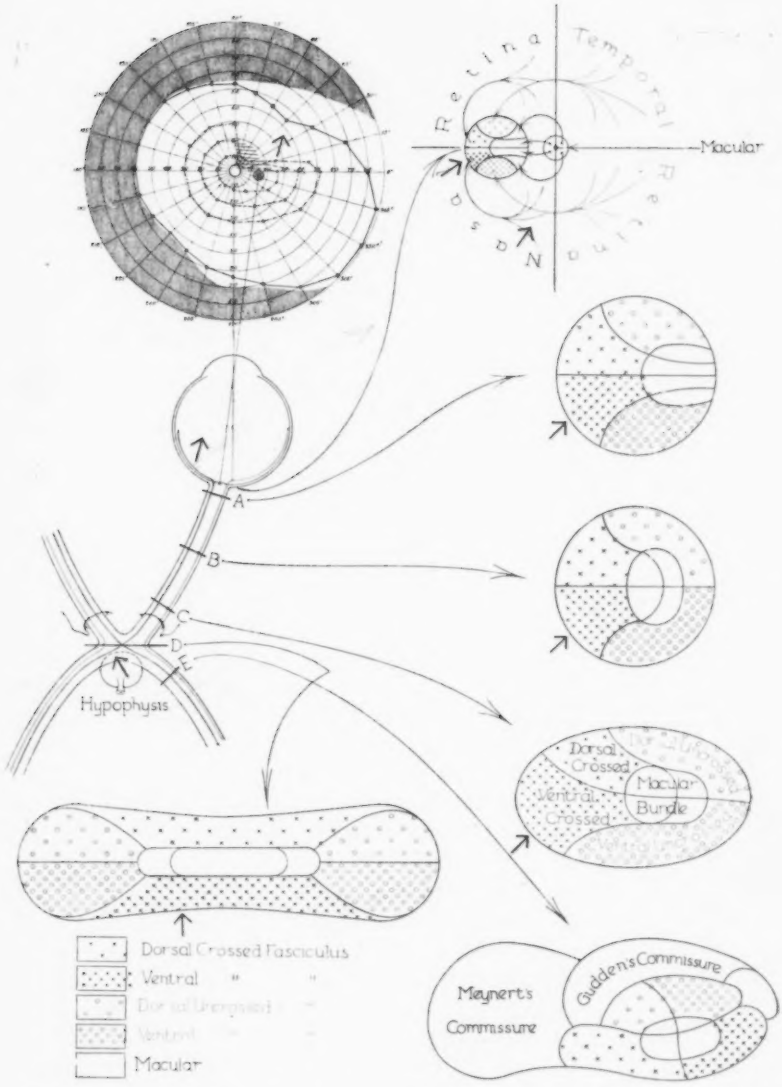

FIG. 1.—Diagram based on Henschen's conception of the disposition in the optic nerve of the several fasciculi. There is indicated an hypothetical involvement of the ventral crossed fasciculus from the right eye, which has produced Stage I. of a right temporal defect (*cf.* perimetric field). The path of the fibres between the point of pressure at the chiasm and the retina is indicated by the short arrows.

The uncrossed bundles (*fasciculi non cruciati*) arise in each eye from the temporal half of the retina, and for the most part retain a lateral position in optic nerve, chiasm and tract as far as the geniculate bodies. In this position they naturally are best protected from the effect of a mesially placed (e.g., retrochiasmal) lesion. Each uncrossed bundle is made up of a dorsal and ventral portion arising from the dorsal and ventral quadrant of the corresponding temporal retina. A bilateral injury of these bundles may, as pointed out in our last paper, give a binasal hemianopsia; a unilateral injury involving one bundle alone, a nasal hemianopsia of the corresponding eye: injury of the ventral fibres alone, an upper quadrantal loss of nasal vision, and so on.

The crossed bundles (*fasciculi cruciati*), likewise composed of dorsal and ventral fibres, arise from the nasal half of each retina, enter the inner (nasal) side of the papilla, pass down the mesial side of the optic nerve, cross at the chiasm and continue down the ventral side of the opposite tract to the contralateral geniculate bodies. These fibres therefore are the most exposed to the effect of a mesially placed lesion, and consequently are the ones most often implicated by hypophysial growths with the resultant bitemporal blindness. Moreover, as the ventral fibres which arise from the lower nasal quadrants remain ventrally disposed throughout their course, when the chiasm is primarily affected by pressure from below, as it is likely to be in the case of a hypophysial tumour, the defect should appear first of all in the upper temporal fields, as illustrated in the diagram. If, on the other hand, the growth is likewise mesial but of infundibular rather than hypophysial origin, the dorsal fibres of the crossed bundle arising from the upper nasal retinal quadrant would be expected to suffer first, with the primary defect showing in the lower temporal quadrant of the field of vision. This, however, is a comparatively rare condition.

The macular fibres (*fasciculus macularis*) leave the retina from the region of central vision about the fovea and enter the temporal side of the optic papilla, separating the dorsal and ventral fibres of the uncrossed fasciculus as they approach the papilla from the temporal retina. From its lateral position as it enters the optic papilla the macular bundle gradually assumes a more central position as it passes down the nerve, where it becomes covered in by the uncrossed fasciculus; though, as a matter of fact, it actually remains somewhat eccentric. It consists, furthermore, of a crossed and uncrossed division—the latter lying in juxtaposition to the uncrossed fasciculus proper, the former crossing at the chiasm together with the crossed fasciculus proper from the nasal retina.

As the macular bundle occupies a somewhat protected position throughout its course, one might conjecture that as a consequence of pressure against nerve, chiasm or tract it would suffer later than the crossed fasciculus in the case of medial pressure and later than the uncrossed fasciculus in the case of lateral pressure. It would appear, however, that the macular fibres are especially vulnerable and, when affected as a consequence of pressure, are

less capable of complete restoration than are the more peripherally disposed fibres.[1]

Types of bitemporal distortion.—Nine possible types or combinations of bitemporal defect, with illustrative cases from the literature, were enumerated some years ago (1897) by Wilbrand in his excellent chapter on "Perimetry and its Clinical Value" in Norris and Oliver's "System of Diseases of the Eye." They were as follows[2]:—

(1) Typical temporal hemianopsia in one eye; in the other the upper and outer quadrant being lost.

(2) Typical temporal hemianopsia in one eye; in the other the lower and outer quadrant being lost.

(3) Only the upper and outer quadrant lost in each eye.

(4) Only the lower and outer quadrant lost in each eye.

(5) Complete blindness in one eye, with temporal blindness in the other.

(6) Complete blindness in one eye, with upper nasal quadrant remaining active in the other.

(7) Complete blindness in one eye with lower nasal quadrant remaining active in the other.

(8) The upper nasal quadrants of both eyes alone remaining active.

(9) The lower nasal quadrants of both eyes alone remaining active. (No reported case.)

In the writers' series, illustrations occur of practically all of these types, and it would be possible, indeed, to add a great number of other combinations to those which Wilbrand has given, particularly if the various hemianopic or quadrantal colour distortions and the sparing or otherwise of the macula in the form or colour field outlines be regarded as types. One might include such field deformations as the following: (1) The sector defects which occur primarily in the colour fields, more often appearing first in the upper temporal quadrants; (2) blindness in one eye with (*a*) a normal field in the other, or (*b*) involvement of only a single quadrant of the nasal retina of the other, or (*c*) with so inconspicuous an upper temporal field defect that it may easily escape detection, or (*d*) a lower temporal field defect of equally slight grade;

[1] In the last paper of this series when we come to discuss the lesions of the geniculo-calcarine pathway it will be pointed out that these divisions into dorsal and ventral bundles for crossed and uncrossed tracts still hold in a measure for the cerebral relay of the visual pathway. That this is particularly true of the temporal loop of this pathway has been clearly shown by Adolph Meyer.

The question of sparing or otherwise of the macula in central versus peripheral lesions must likewise be deferred to a later paper.

[2] Lest there be some confusion it may be said that throughout this paper when we speak of a nasal or temporal hemianopsia we refer to that half of the visual field and not to the retina itself.

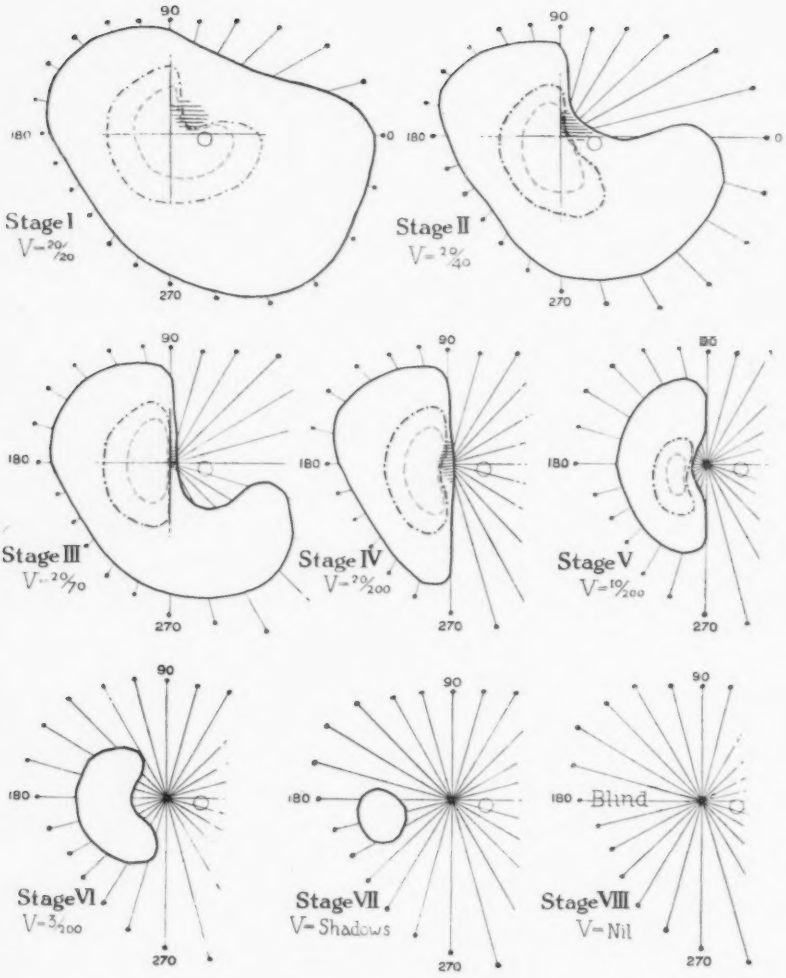

FIG. 2.—Showing the eight stages of a progressing right temporal defect.

(3) blindness in one eye with superior hemianopsia in the other, the ventral crossed and uncrossed fasciculi being completely blocked, whereas the dorsal fibres are largely spared. Thus, were classifications of this sort worth recording, an infinite number of types of increasing complexity might be given. Wilbrand himself states that "the fields of vision found in diseases of the chiasm afford only a glance, as it were, into the momentary condition of the path."

Hence it seems to us far more preferable to look upon the perimetric deviations as stages rather than types, particularly as the condition in the two eyes rarely progresses symmetrically, and we have consequently arranged the cases from this viewpoint.

Stages of Temporal Hemianopsia.

The periods in an advancing process which we have come to speak of as "stages" we have enumerated serially, from the normal field to the stage of blindness. These steps, or stages, have been selected out of the many possible ones for the reason that the larger number of our cases happen to have been caught in them, and not with the idea that the advancing process tends to halt at these separate stages, except possibly in Stage IV. In the following description of these stages (cf. fig. 2) the average measurement of vision estimated from the tests on eyes which in our perimetric records accord with the stage in question, is also given.

Stage I shows merely an upper temporal quadrantal defect in the colour fields, often with a slight slanting off of the form fields in the same quadrant. At this time a paracentral relative or absolute scotoma not infrequently may also be made out in the upper temporal quadrant. In this stage the patient usually has central vision of $\frac{20}{20}$, but may be conscious of a slight central haziness which, however, is difficult of objective demonstration with ordinary methods of perimetry.

Stage II.—The defect in the upper temporal quadrant, anticipated by the slant in the form field of Stage I, becomes realized and the colour fields recede considerably in the lower temporal quadrant. The paracentral scotoma persists in whatever may be left of the quadrant, and in patients with good powers of observation the limits of a relative central scotoma may occasionally be outlined. Central vision falls to an average of $\frac{20}{40}$.

Stage III.—Here complete temporal hemiachromatopsia is usually present for smaller discs, but for larger sized discs colour vision can often still be demonstrated across the mid-line in the lower quadrant. In passing into this stage it is notable that the temporal form field sinks below the horizontal line, particularly in the region of the blind spot, after the manner of an enlargement.

There is also a peripheral contraction, so that the resulting outline is variably gourd-shaped.

The relative central scotoma is present, and a hemiopic pupillary reaction is usually demonstrable by the rotary shutter test. Central vision becomes $\frac{20}{50}$ to $\frac{20}{70}$.

Stage IV.—Hemianopsia has become complete for form and colour, the macula often being spared by the otherwise vertical meridian.

The relative central scotoma increases in size for form and becomes absolute for colour, green usually being lost before red and red before blue. Vision stands at $\frac{20}{70}$ to $\frac{20}{200}$.

Stage V.—At this stage there is definite constriction of the remaining hemianopic colour and form fields, and the latter may begin to show constriction either above or below, but more often below.

The relative central scotoma becomes absolute, and eccentric vision is about $\frac{15}{200}$.

Stage VI.—Colour vision is lost except occasionally for large discs. The nasal form field begins to shrink away from the vertical meridian.

Central vision is nil, and eccentric vision reduced to the neighbourhood of $\frac{5}{200}$.

Stage VII.—A small area is left in the nasal field, which can be barely outlined with large discs or with small flash-lights. Vision is reduced to shadows or light perception.

Stage VIII.—Blindness. Pupillary reactions nil.

The least affected eye in the meantime is progressing along the same route, for it cannot be too strongly emphasized that the process rarely advances with equal steps in the two eyes. Thus we may see Stage VIII—namely, of blindness—in one eye when the other has not passed beyond Stage I or Stage II, or has retrogressed to Stage II or Stage I, or indeed may have become normal. It is important to appreciate, moreover, that there are probably recessions and advances in the process from time to time, though the general tendency of the condition, unoperated, is toward an advance. It is quite possible that in this way the macular fibres may become involved, and also that they are somewhat more vulnerable than the other fibres, so that after an advance up to the point at which the macular bundle is involved, even though there is a recession in the process with widening of the fields, a relative scotoma of the fibres of the macular bundle may be left in the involved quadrant (cf. Case 3). This may possibly account for the presence of these scotomas, which Bartels, in Germany, and de Schweinitz and Halloway [12] in this country were the first to describe in detail, and we have been able to corroborate their findings in a great number of our cases. Another explanation has been offered by

J. Herbert Fisher [13], who suggests that the process is the result of traction upon, rather than of pressure against, the chiasm, and that the macular fibres, owing to their highly specialized function, may suffer more than the others. It would seem to us that the two conditions, pressure and traction, must go hand in hand; and as a matter of fact we have come to believe from our *post-mortem* studies that in most cases the tracts suffer far more than the optic nerves or chiasm, for they may be thinned out by pressure into mere ribbons even when the chiasm shows relatively little deformation.

It is quite apparent that in the majority of these cases the upper fields suffer more than the lower,[1] and this is apt to be so even in the advanced stages, for there are a number of examples in the series in which merely a small patch of vision remained in the lower nasal fields, where shadows of large discs were still perceived. Moreover in one or two cases a small patch of vision has returned after operation in the lower nasal quadrant of a previously blind eye (cf. Case 6). In one case, too, there was a restoration of almost the entire lower field for form, suggesting a form of superior hemianopsia, the crossed and uncrossed dorsal fasciculi of the eye in question having escaped from complete destruction. Another interesting fact to which attention has already been called is that in the progressive constriction of the fields from Stage III to Stage IV—namely, from the gourd-shaped field to that of exact hemianopsia—a small point representing the tip of the gourd may remain isolated in the temporal field. Under these circumstances, with return of vision the field widens out from the lower zone and soon envelops this isolated patch (cf. Cases 5, 6 and 7).

The fact that the macular fibres may suffer most, as already suggested, is apparent also in cases in which after Stage IV has been reached and a successful operation has been performed, with subsequent widening of the fields to their normal peripheries, a relative central scotoma persists long after normal field peripheries have been regained (e.g., Case 3). When central vision is actually lost, in cases which have advanced beyond Stage IV, perimetric observations of any exactitude become impossible unless two points of eccentric fixation are employed.

Some of these stages can be well illustrated by giving a more or less complete series of charts from an individual history, for in some patients circumstances have permitted us to follow the condition over a long

[1] To this also J. Herbert Fisher [14] has called attention.

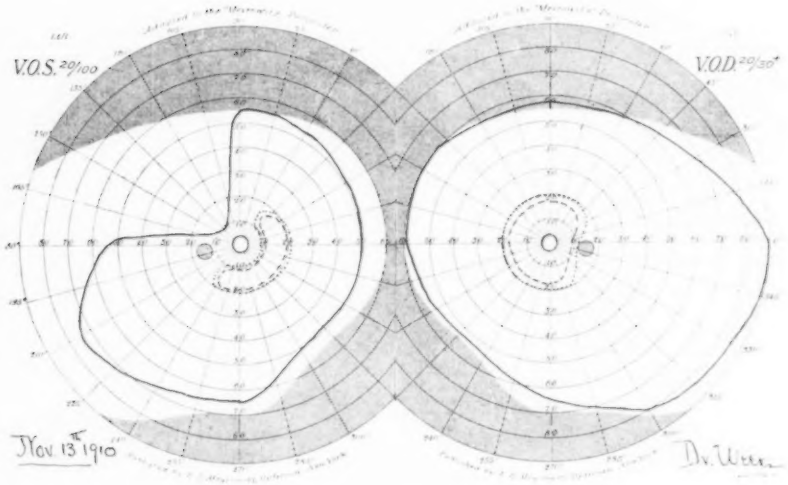

FIG. 3.—Case 1. Fields of November 13, 1910 (Dr. Weeks), fourteen months before operation. O.S., Stage II.

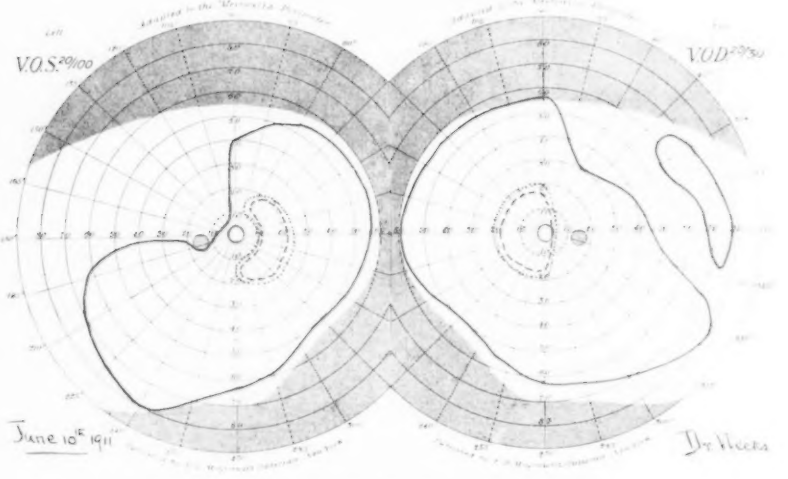

FIG. 4.—Case 1. Fields of June 10, 1911 (Dr. Weeks), seven months before operation. O.S., Stage II +; O.D., Stage I.

period, so that we may have as many as twenty serial perimetric observations. In the following case, for example, our own charts were fortunately supplemented by observations taken elsewhere before the patient came under our care.

Case 1 (J. H. H., Surg. No. 29,117).—*Dyspituitarism, enlarged sella, bitemporal hemianopsia, operation, restoration of vision.*

January 20, 1912.—Admission of Miss E. V. H., aged 54; referred by Dr. John E. Weeks, of New York. Slight symptoms of dyspituitarism, with incipient acromegalic manifestations. The cranial X-ray shows a greatly distended sella. No headaches, nausea or vomiting; always strong and well.

Vision began to fail eighteen months ago. Four months later an opaque blur was observed in the upper left temporal quadrant; and gradually the entire lateral vision in the left eye was lost. Several months before admission a similar blur appeared in the upper right temporal quadrant, but this has not, as yet, advanced as far as on the left. Examinations by Dr. Weeks during this period verified these subjective symptoms (cf. figs. 3, 4, and 5, for November 13, 1910, June 10, 1911, and January 11, 1912). Vision decreased meanwhile to V.O.S. $\frac{20}{200}$, V.O.D. $\frac{2}{40}$. The process has been advancing with rapidity during the past few weeks.

Examination of eyes.—No diplopia, pupils equal and active to light, accommodation and consensually. Movements complete; no strabismus, nystagmus or von Graefe's sign. *Fundi:* O.D., optic disc shows a slightly heightened pallor of the nasal side, slight suggestion of arterio-sclerosis; otherwise fundus normal. Lamina cribrosa sharp. No swelling of disc. O.S. has the same appearance, with slightly greater pallor of the disc. Pupillary reaction roughly seems better from the nasal than from the temporal retinæ. Wilbrand test negative. Pupillary reactions to the small lights of Hess (objectively with Krusius type of pupillometer) show no better response from the nasal than from the temporal retinæ; patient could not use entopic method.[1] V.O.S. $\frac{10}{200}$, V.O.D. $\frac{20}{50}$.

Fields (fig. 6) show a bitemporal hemianopsia with the macula bisected on the right (Stage IV) and lost on the left (Stage V).

January 24.—*Fields* and acuity unaltered.

January 27.—*Transphenoidal operation.* Partial removal of struma. Perfect recovery, with subjective improvement in vision on the tenth day.

January 31.—*Fields* of this date show practically no change to small discs, and acuity remains unaltered.

February 9.—*Fields* show improvement; now almost typical bitemporal hemianopsia, the macular defect on the left being somewhat less extensive. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{20}{40}$—.

[1] Could this patient have been examined, as have been more recent ones, with a rotary shutter and strong illumination a hemiopic pupillary reaction would doubtless have been demonstrable. (*Tr. Ophth. Sect. Amer. Med. Assoc.*, June 1914, pp. 348-353.)

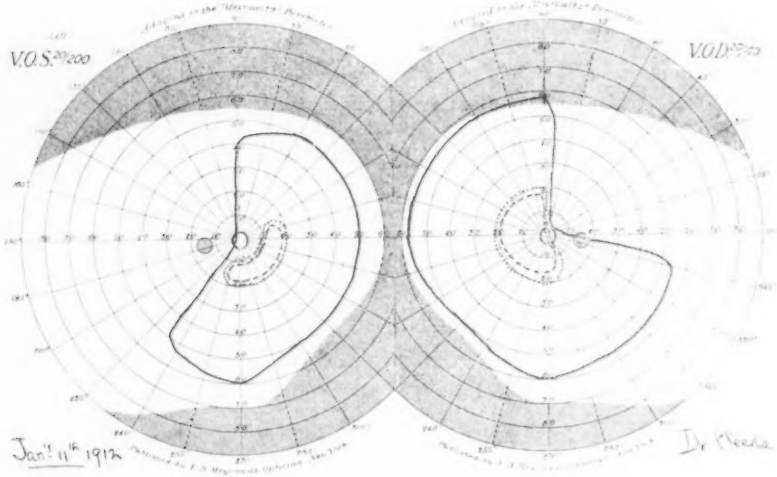

FIG. 5.—Case 1. Fields of January 11, 1912 (Dr. Weeks), sixteen days before operation. O.S. Stage III +; O.D. Stage II.

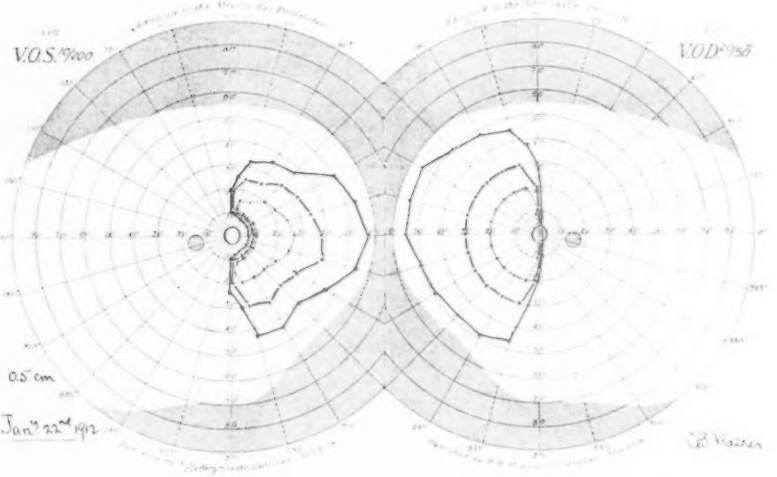

FIG. 6.—Case 1. Fields on admission, with 0·5 cm. discs, five days before operation. O.S., Stage V; O.D., Stage IV.

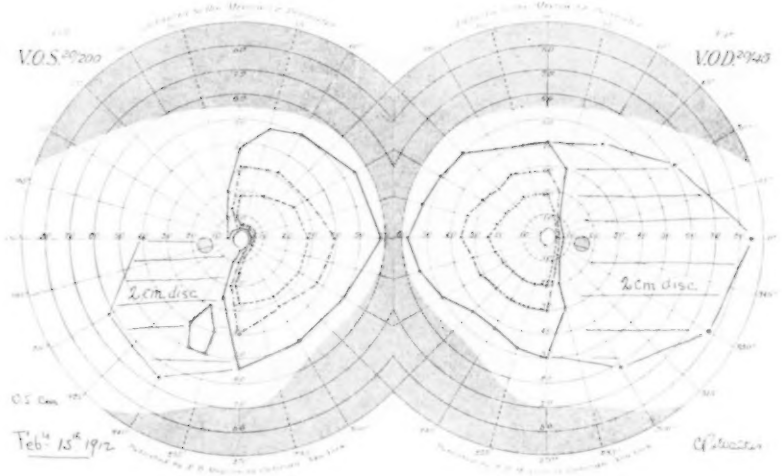

FIG. 7.—Case 1. Fields of February 15, 1912, nineteen days after operation, shaded areas to 2 cm. discs, showing marked recession.

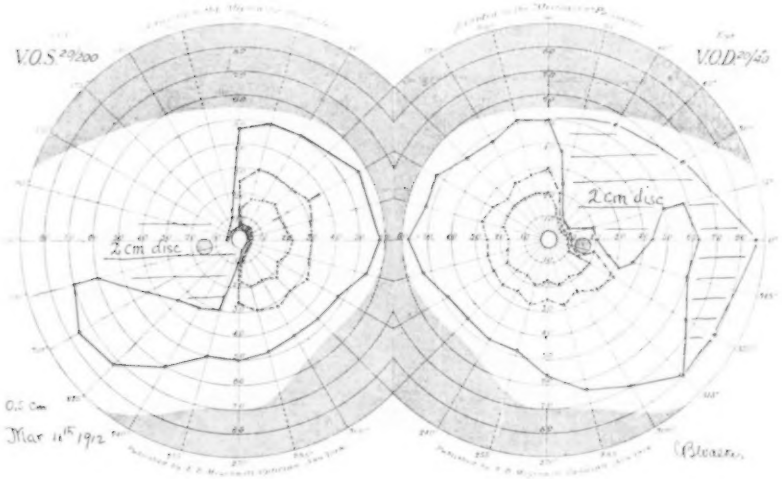

FIG. 8.—Case 1. Fields of March 11, 1912, six weeks after operation: shaded areas to 2 cm. discs. Showing recession O.S. to Stage III; O.D. to Stage II.

February 15.—*Fundi* unchanged. *Fields* (fig. 7) show marked improvement, with return of vision to 2 cm. discs in the whole right temporal field and lower left temporal field. V.O.S. $\frac{20}{200}-$, V.O.D. $\frac{20}{40}$. Patient discharged.

March 11.—*Returns for observation. Fundi:* O.U. unchanged. Fields (fig. 8) show further improvement: recession on the right to about Stage II, and on the left to about Stage III. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{20}{40}$.

April 5.—Report from Dr. Weeks. Fields (fig. 9) appear normal on the right; left in Stage II (?). V.O.S. $\frac{20}{200}$, V.O.D. $\frac{20}{25}+$.

May 20.—*Returns for observation. Fundi:* O.D. normal except for very slight arterio-sclerosis and rather pale disc; O.S. disc paler than on the right but appearance otherwise the same. *Fields* (fig. 10) normal on the right and on the left approaching Stage II, with central scotoma. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{20}{25}$.

July 22.—Reports by letter "almost perfect vision in both eyes."

September 18.—*Reports in person.* Fields (fig. 11), plotted for the first time with graduated discs, show more clearly than those of *May* 20 the character of the improvement on the left. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{20}{25}$.

January 5, 1913.—*Reports in person. Fields* (fig. 12), the right remains normal and the left shows further improvement, but the central scotoma persists. V.O.S. $\frac{20}{100}$, V.O.D. $\frac{20}{15}$.

Comment.—This story illustrates in sufficiently typical fashion not only the manner in which the field defects usually advance, but also the manner in which recessions in the defects occur after the nerves have been relieved from pressure. It will be noted that the first charts taken by Dr. Weeks, on November 13, 1910 (fig. 3), showed our Stage II advancing to Stage III in the left eye, with an evident involvement of the ventral crossed fasciculus to that eye. It is quite probable that at that time more detailed tests would have shown a relative central scotoma, in view of the fact that the colour fields are so markedly constricted. The right eye at the same time was practically normal, or possibly in a latent condition of Stage I, for the evident constriction of the temporal colour fields shows that some change has occurred. Dr. Weeks's two subsequent charts (figs. 4 and 5) of seven and fourteen months later show a progressive advance in the left eye to Stage III and in the right to Stage II, and during the eleven days intervening between his last observation and the patient's admission there was a rapid advance to Stage V in the left and Stage IV in the right eye. After the operation there was a fairly prompt improvement on the right with return to normal from Stage IV in three months, and a slower but progressive return on the left from Stage V to Stage II during the year after the operation, which in all likelihood represents the high-water mark of possible restoration. As a matter of fact the

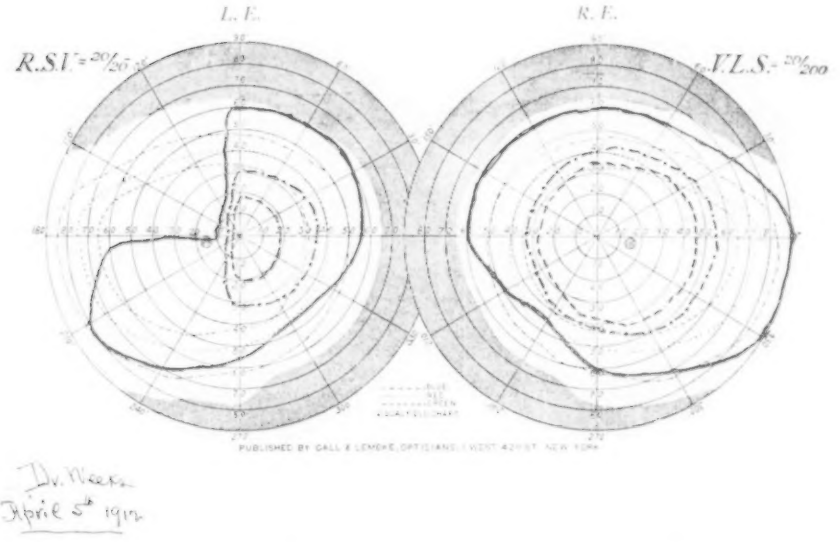

FIG. 9.—Case 1. Fields of April 5, 1912 (Dr. Weeks), ten weeks after operation.

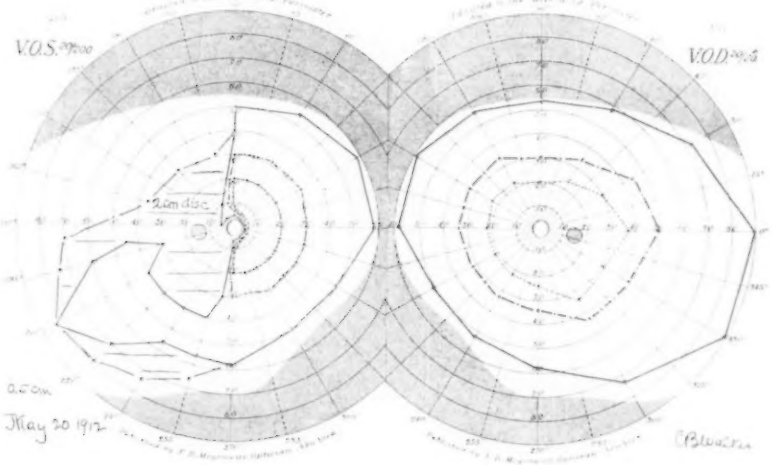

FIG. 10.—Case 1. Fields of May 20, 1912, sixteen weeks after operation: shaded area to 2 cm. disc. O.S. Stage III −; O.D., normal.

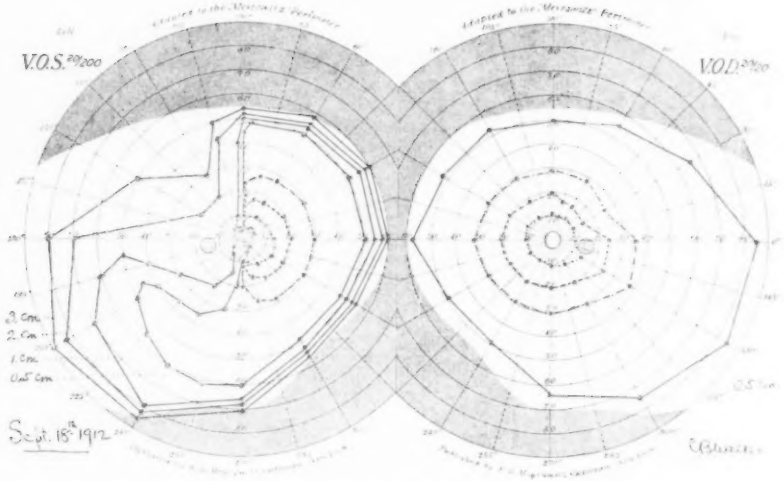

FIG. 11.—Case 1. Fields of September 18, 1912, by improved method with graded discs, eight months after operation. O.S., Stage III – ; O.D., normal.

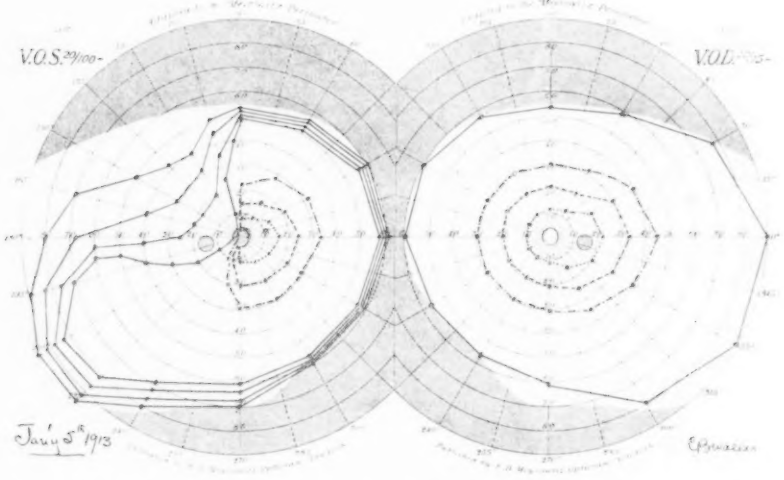

FIG. 12.—Case 1. Fields of January 5, 1913, one year after operation, showing further improvement. O.S., Stage II; O.D. remains normal.

improvement was much less rapid than is usually the case after a successful operation of this type when the process has not advanced beyond Stage IV. It is customary for us to see a much more striking restoration during the first few weeks, as will be apparent from some of the subsequent histories.

The case has been selected particularly for the reason that it illustrates the progressive improvement in the technique of making these observations, for in the fields of February 15 (fig. 7), March 11 (fig. 8) and May 20 (fig. 10), taken by ourselves after the operation, the outlines were plotted with discs of 0·5 cm. diameter, and we merely indicated by lines upon the charts that some vision to larger (2 cm.) discs was apparent in the previously blind temporal fields. As will appear from the observations of September 18, 1912 (fig. 11), and January 5, 1913 (fig. 12), we had by this time begun to record upon the same chart the outlines given by discs of graduated areas, from $\frac{1}{4}$ sq. cm. to 16 sq. cm.—an extension of the screen method of Bjerrum [2] [3] [4] and Rönne [17] [18] [19] used by them in cases of glaucoma and optic atrophy, adopted for our purposes to the perimeter.

Attention may be called to the fact that the earlier charts, not only our own but those of Dr. Weeks, failed to indicate a relative central scotoma, which in the light of subsequent experiences was in all probability present. Thus the field of the right eye, taken January 22 (fig. 6), should in all probability not have shown the macula clearly bisected, but should have indicated a relative central haziness, the existence of which, at the time, is reasonably assured by the lowered $\frac{20}{50}-$ vision.

It is occasionally indicated, even on these older and less carefully plotted charts (as shown in fig. 7), the patches of vision may be retained in the blind fields, or that vision in these fields may first return in patches—a matter which will be entered into more fully later on. Suffice it to say, we did not appreciate the significance of these islands until we came regularly to employ the graded discs.

Technique of Perimetric Examinations (C. B. Walker).

Though the methods of plotting fields which we have come to depend upon are applicable to all cases, the information to be gained therefrom is much greater in the group of anterior (chiasmal) lesions, to which these hypophysial cases belong, than in the cases of field defects due to central (cerebral) lesions. Thus if we make a primary

division of fields into (1) those taken in cases of anterior lesions and (2) those taken in cases of posterior lesions it is notable that the line of cleavage between blind and seeing field is generally much sharper in posterior than in the anterior cases. For instance, it is the rule to find that a patient with homonymous hemianopsia of posterior origin fails to note the motion of objects, no matter how large, to the blind side of the fixation point until one edge projects into the seeing field, while a patient with an hemianopsia of anterior origin who shows a fairly sharp line of cleavage for small form and colour discs, may still be able to detect gross movements on the defective side of the field.

The representation on charts of the full functional capacity of the retina in these cases was not satisfactory with the ordinary equipment, on account of lack of uniformity. Accordingly a new series of discs was devised, which ranged in graduated sizes above and below the ordinary normal or 0·5 cm. disc. By the use of such a series we hoped to be able to trace fluctuations in the visual fields more accurately, and possibly get an index from the isopters of the different discs as to what the next stage would probably be.

In choosing a basis for our disc series it did not seem advisable to depart too radically from the system in general use, since most of the patients came from a distance with several previous fields indicating a progression of stages, and on their return home continued to have their fields plotted on the original basis. This serial method devised by one of us may be used both in the general manner with the perimeter and on the Bjerrum screen; but with the latter method the denomination of the visual angle fraction must be changed, since practically all the perimeters in this country are built on a radius of about 280 mm.

Our method, then, elaborated with especial reference to hypophysial cases and other progressive lesions, does not involve the use of a special perimeter, but the discs cover a considerably larger range, the largest being of such a diameter that further increase in size gives practically no added information in the defective areas. In order to have the test objects symmetrically disposed about a central point from which the measurements are taken and which lies as nearly as possible in the same focal plane as the fixation point, circular, flat discs are used, having only a knife edge, serving merely to protect the mat surface of the disc. The rimless feature of these discs is essential, since we have found, especially with small test objects with relatively large rims and handles, that motions of the carrier may be reported before the

test object itself is noted. With rimless discs and wire handles, even without repeated warnings to the patients, we are much more confident that reported motions are actually due to the test object.

Further, since the circle having the same diameter as the side of a given square has an area only three-quarters the area of the square, our discs are mentioned in terms of circular areas, in order to avoid confusion as to whether a square or circular test object has been used.

In regard to the size of the discs, it has been found that diameter ratios of 1 : 2, or what is the same thing, area ratios of 1 : 4, give readings most satisfactorily charted. The normal or 5 mm. disc being so commonly used, must be included in the series, and is therefore taken as a starting point. Above and below this size, the discs range in diameter ratio of 1 : 2. In all the charts herein presented, where four form peripheries are shown, the inside line always represents, unless otherwise stated, the normal disc, and the outer lines discs of increasing size in the given ratio.

On the back of each white disc is a green disc, and at the other end of the wire handles and at right angles to the white and green discs is another double disc of red and blue. This arrangement prevents the patient from seeing more than one disc colour at a time, and enables the observer to change the colours without the patient's being aware of his doing so.

It has not been found necessary to vary the testing apparatus from that usually employed, and mechanical perimeters have not been found to offer any advantage over the plain perimeter in taking fields as completely, accurately and rapidly as possible. It may be said that a special blinder is also used, fitted with two attachments by which a field may be taken on an eye having a central scotoma, if vision and fixation are good in the other eye.[1]

There are certain theoretical considerations of interest in relation to the distribution of the fibres damaged by the pressure and of their terminals in the retina. Thus if it be assumed that when a visual nerve-fibre is subjected to pressure in any part of its path its function is first weakened before becoming entirely blocked, one may conceive, in the case of pressure against a bundle of fibres, of concentric zones of diminishing effect of the pressure. In the first zone would be found fibres entirely blocked or even destroyed; in the second zone some scattered normally functionating fibres might appear; in the third

[1] A more detailed description of these instruments may be found in an article recently published by one of us [20].

occasional entirely blocked or extremely depressed fibres might be found among those considerably depressed but still imperfectly functionating; outside of these zones a fourth zone of slightly depressed fibres would be located, and so on. If such zones were present in the nerve it would not be surprising if they disclosed themselves in the retina as zones of different stimulability, that is, if a defect in the form field were demonstrable with the normal sized disc, the defect recorded by a larger disc would be still smaller, and the distance between the two lines in the normal field would represent practically the difference in the disc radii, while in the defective region the lines would spread considerably apart, depending on the nature and degree of damage in the postulated zones.

A study of the fields before and after operation gives considerable evidence that atrophy of the visual fibres does not necessarily take place until they have been under pressure sufficient to block their function for six months or a year, or even longer. If a sharp atrophy or destruction of visual fibres has taken place no response to graduated discs in the defective area is expected, the larger discs being perceived only when their margins reach the line of cleavage between blind and seeing fields. In such visual defects there is obviously little hope of improvement. Altogether, then, field examinations in conditions and with changes of this sort would possibly not only add considerable information in regard to the functional capacity of the optic tract and retina, but would also, when a series of fields and time durations were available, give some evidence of the position, size and rate of growth of the lesion.

The method of Bjerrum involves the use of a black screen 2 metres square, at a distance of 2 metres from the patient. The test objects are spots 3 mm. and 6 mm. in diameter. At a distance of 2 metres the field for these test objects, although only 30° to 40°, may be considerably larger than the screen. Therefore, in order to confine the measurements to the area of the screen, the fixation point must be changed from point to point. To record the field reading on the chart the measurements of meridian and arc, ordinarily made direct from the perimeter, must now be actually measured on the screen for each reading with rule and protractor. The test object is recorded in terms of a factor which represents its visual angle, that is, $\frac{\text{diameter of disc}}{\text{distance from patient}}$, since this fraction multiplied by the factor $\frac{180}{\pi}$ gives the visual angle in degrees.

This method was adopted on account of the difficulty of making accurate discs below 1 mm. in diameter. If such discs could be made so as to represent visual angles of five minutes (about equal to $\frac{20}{20}$ vision) and less on the ordinary perimeter, several objections to the Bjerrum method would be obviated. These objections are, first, that the discs have a variable visual angle and focal plane as they are used at various points on the flat screen surface; second, that presbyopic patients cannot use their glasses to advantage for the middle distance of 2 metres, since they are made for 6 metres or for reading; third, the loss of illumination intensity due to the instance; and finally and most important, the readings are so laboriously made and recorded that as the patient grows fatigued results become unreliable.

We have been able to make discs of very small size, extending our series downward until the visual angle on the ordinary perimeter approaches one minute, very close to the limit of visibility. As was anticipated, the same results are obtained by this method as with the Bjerrum method, but with greater facility and rapidity. The area ratio is maintained at 1 : 4 throughout.

Thus we have devised similar methods of examining the areas both outside and inside the boundaries established by the normal disc. The larger discs are especially useful in giving added information concerning the residual functional capacity in a defect found by the normal disc. If this defect is of considerable extent the very small discs may not give much added information concerning the functional capacity of the remaining field, but it is when there is practically no defect to the usual discs that the very small discs may be found to give the most valuable, sometimes most unexpected information.

Case Reports illustrating Stages of Bitemporal Hemianopsia.

The first draft of this paper was drawn up two years ago with the selection of twenty illustrative case reports from our Baltimore series. Significant of the improvement in our technical methods of perimetry is the fact that though several of these earlier cases are of unusual interest we, nevertheless, in this interval have come to supplant them, one by one, with more recent cases as inaccuracies in our former methods became apparent. Possibly, should we delay for another two years we might have the same feeling in regard to our present charts. However, the following thirteen records which we have finally chosen may

serve as fairly satisfactory examples of the conditions to which we wish to call attention.[1]

The cases will be divided into two groups: (1) those in which the hemianopsia was produced by a lesion, usually a glandular struma so-called, originating within the sella—a condition favourable for a transphenoidal operation; and (2) those in which it was produced by a suprasellar tumour—which does not necessarily deform the sella turcica, whatever it may do to the chiasmal cross-roads themselves.

(*a*) *The intrasellar tumours causing bitemporal hemianopsia.*—It will be observed in the following nine case reports (Case 2 to Case 10) that an attempt is made to present the cases serially so that one or the other eye at the time of the earliest observation is at a stage slightly in advance of that in the preceding case. In the following example (Case 2) when the patient was first seen the fields were regarded as normal, and consequently the case is one of the several that are not included in the given statistics, which, as has been told, were based on the conditions found at the patient's first admission. It is an illustration of the earliest field deformation which we have come to recognize as typical.[2]

Case 2 (P.B.B.H., Surg. No. 1484).—*Acromegaly with early unilateral field defect in Stage I. Operation. Improvement.*

November 15, 1913.—Admission of Miss E. A. C., a school teacher, aged 35, referred to by Dr. H. B. Hawley, of Syracuse, N.Y. Typical early acromegaly, with enlarged sella, headaches, amenorrhœa, asthenia and ocular fatigue.

Examination of eyes.—Fundi, fields, acuity, &c., show no abnormality.

July 10, 1914.—*Readmission*, owing to advance in symptoms. *Fields* (fig. 13): Left eye shows Stage I, with relative paracentral scotoma and lowering of acuity to $\frac{20}{50}$; right normal.

July 16.—*Transphenoidal operation.* Partial removal of tense chromophile struma. Immediate subjective improvement; discharged in ten days with V.O.S. $\frac{20}{25}$, but no change in field peripheries when they were taken on *July* 22.

August 26.—*Returns for examination.* Great improvement in all respects, with rapid diminution of the pituitary myxœdema. The left field shows widening of all peripheries, though there is still a slight slant of the upper temporal area. There is no trace of the scotoma even to minute discs. V.O.S. $\frac{20}{20}$, V.O.D. $\frac{20}{15}$.

[1] In the case records used in this report, those from the Baltimore series are given their J.H.H. surgical number, and we wish to express our obligation to the Johns Hopkins Hospital for the privilege of including them here.

[2] It may be said that in the succeeding charts the colour peripheries are (unless otherwise mentioned) always plotted to the normal test object; and that a single outline for the form field is also taken with the same sized disc. When the graded discs are used the peripheries from within outward represent, serially, registrations from $\frac{1}{2}$, 1, 2, and 4 cm. discs.

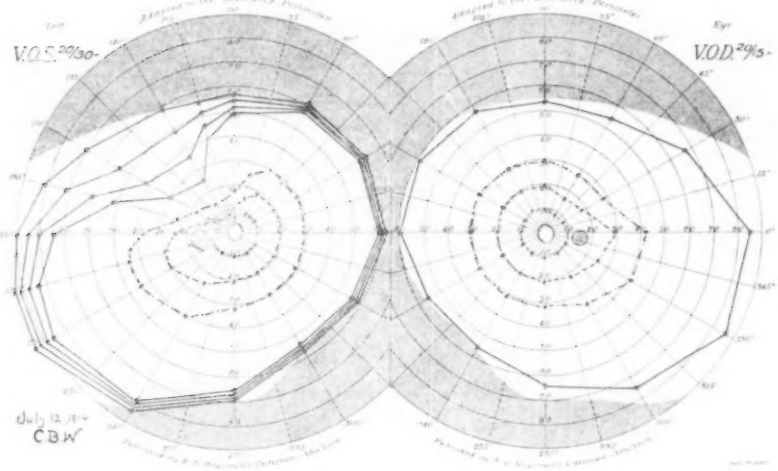

FIG. 13.—Case 2. Fields of July 12, 1914, showing on the left the typical outlines of the form field of Stage I, as plotted by the four graduated discs ¼ sq. cm. to 16 sq. cm.

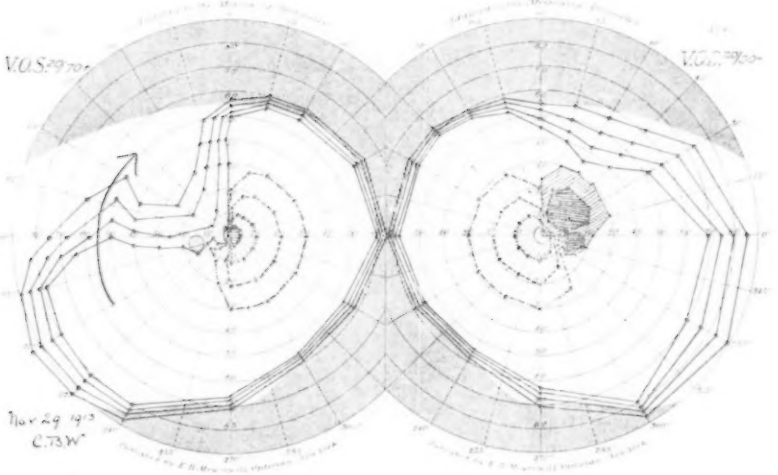

FIG. 14.—Case 3. Fields of November 29, 1913, three days before operation. Advanced Stage II on left, and advanced Stage I on right. In left field arrow shows direction of post-operative recession with envelopment of what will be the subsequent scotoma (cf. fig. 15).

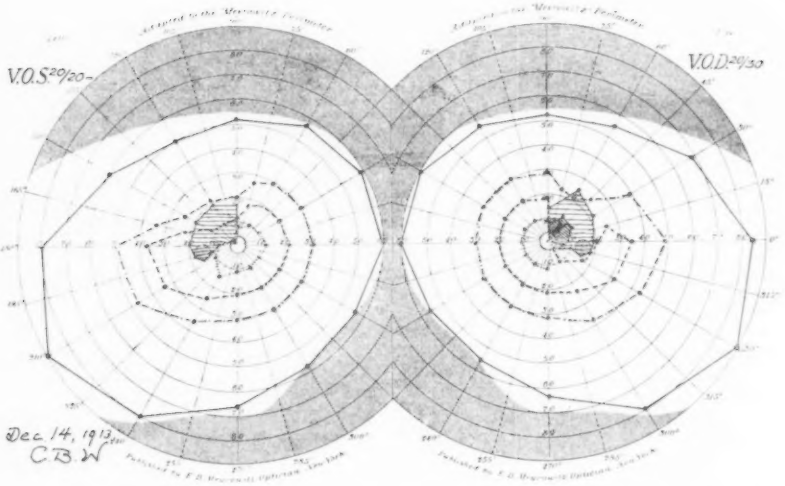

FIG. 15.—Case 3. Fields of December 14, 1913, twelve days after operation, showing recession to early Stage I, with persistent relative scotomata.

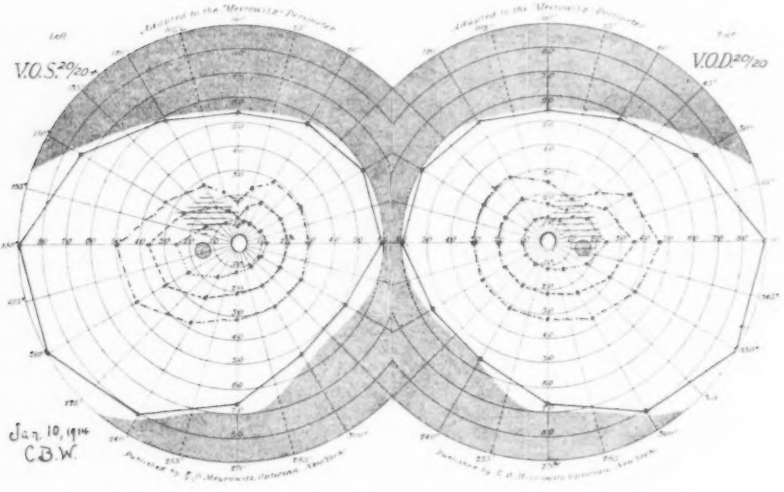

FIG. 16.—Case 3. Fields of January 10, 1914, six weeks after operation. Practical return to normal.

Comment.—In view of the normal right eye, one cannot be certain of course, in such a case that the condition might not have eventually proved to be an homonymous one. There have been two or three very similar cases in the series.

The following example shows an advance to Stage II at the time of admission.

Case 3 (P.B.B.H., Surg. No. 1135).—*Acromegaly with field defects approaching Stage II. Operation with recession to normal.*

November 26, 1913.—Admission of H. A. E., aged 23, a signal-tower operator, referred by Dr. W. O. Paul, of Dorchester, Mass. Typical early acromegaly with faulty vision for five or six months, headaches, enlarged sella, &c.

Examination of eyes.—Pupils show practically normal reactions. No nystagmus, exophthalmos, von Graefe, or limitation in movements. *Fundus:* O.U. Optic disc pale but with a slight blush of injection present. Nasal side of disc is paler than normal but still has more colour than temporal side. Margins slightly blurred by a low grade of œdema and some injection. Very slight venous congestion. Fundus otherwise clear. *Fields* (fig. 14) show an advanced Stage II on the left, and on the right an advanced Stage I with practical hemiachromatopsia, an upper temporal slant of the form fields and a paracentral scotoma in the quadrant. V.O.S. $\frac{20}{70}$, V.O.D. $\frac{20}{30}$ −.

December 2.—*Operation.* Usual transphenoidal procedure with partial removal of large chromophobe struma. Uneventful recovery with prompt subjective improvement in vision.

December 14.—Fundus has lost its injection. *Fields* (fig. 15) show restoration, both right and left, to early Stage I. V.O.S. $\frac{20}{20}$ −, V.O.D. $\frac{20}{30}$ −. *Discharged.*

January 10, 1914.—Reports for examination. *Fields* (fig. 16) practically normal though a slight trace of the scotomata is still demonstrable. V.O.S. $\frac{20}{20}$ +, V.O.D. $\frac{20}{20}$.

January 20.—Has returned to his post as a signalman, on night duty, having successfully passed the imposed rigorous tests for vision.

Comment.—The charts of this and of some of the immediately following cases indicate how it is that a greatly enlarged blind spot or a central scotoma persists after the closing in of the form fields, which spread upward in the direction of the arrow (fig. 14) and fuse around the paracentral area where the fibres seem to suffer most.

The following case is an example of post-operative recession in a patient admitted in Stage III.

Case 4 (P.B.B.H., Surg. No. 972).—*Hypopituitarism with optic atrophy and field defects in an advanced Stage III. Operation with recession to Stage I.*

February 28, 1914.—Admission of Carl S., aged 49, a watchmaker, referred

by Dr. A. Ziegler, of Plainfield, N. J. A typical example of pituitary insufficiency, with large eroded sella, adiposity, headaches and subjective failure of vision for eight months, beginning in the left eye and two months later in the right. Progressive failure.

Examination of eyes.—Slight exophthalmos. Pupils show normal reactions. *Fundus:* Temporal portion of discs paler than normal, but might not be noted on casual examination. *Fields* (fig. 17) show typical and fairly symmetrical advanced Stage III, which particularly on the left and to the smallest disc approaches Stage IV. V.O.S. $\frac{20}{100}$ −, V.O.D. $\frac{20}{50}$ +.

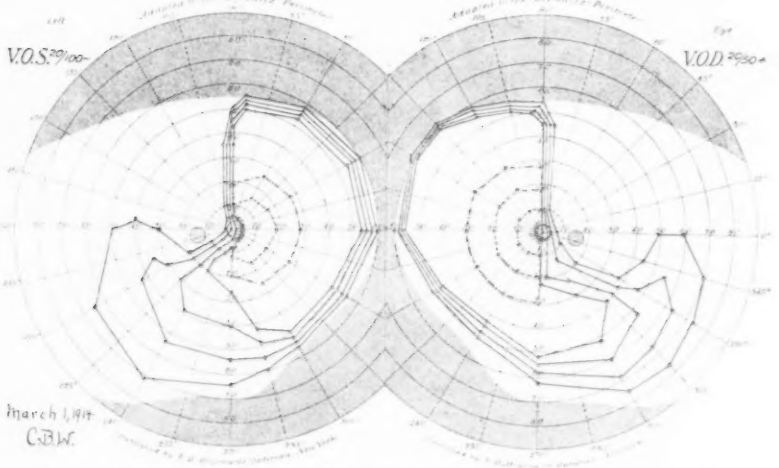

FIG. 17.—Case 4. Fields of March 1, 1914, four days before operation. To the smaller discs the condition is seen to be almost in Stage IV with vertical meridian.

March 5.—Operation. Usual transphenoidal approach with extensive extirpation of soft chromophobe struma. Uneventful recovery with prompt subjective improvement in vision.

March 10.—Fields (fig. 18) are widening to Stage III on the left and Stage II on the right. V.O.S. $\frac{20}{50}$ +, V.O.D. $\frac{20}{30}$.

March 16.—Fields show further improvement (fig. 19) with fusion of circumferential peripheries to 1 sq. cm. discs. V.O.S. $\frac{20}{30}$ −; V.O.D. $\frac{20}{20}$ −. Discharged.

Comment.—Of this typical retrogression in cases no further advanced than Stage III there have been several examples in the series. Though at the time of the patient's discharge eleven days after the operation, the temporal peripheries (fig. 19) had not fused to the $\frac{1}{4}$ sq. cm. (that

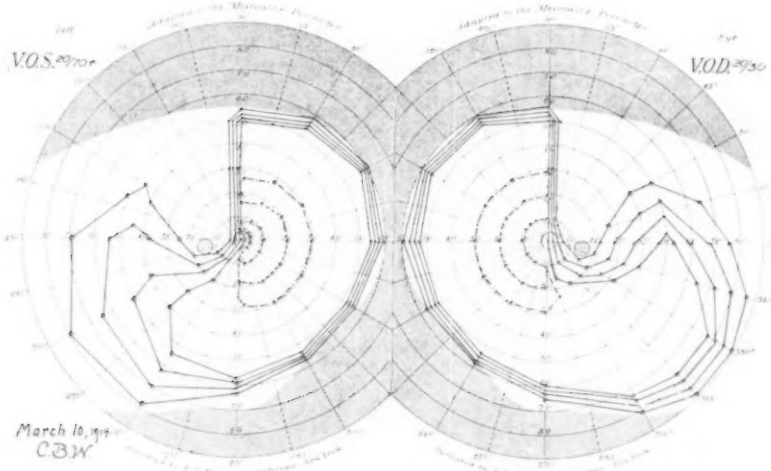

FIG. 18.—Case 4. Fields of March 10, 1914, five days after operation, showing recession to Stage III on the left, and toward Stage II on the right.

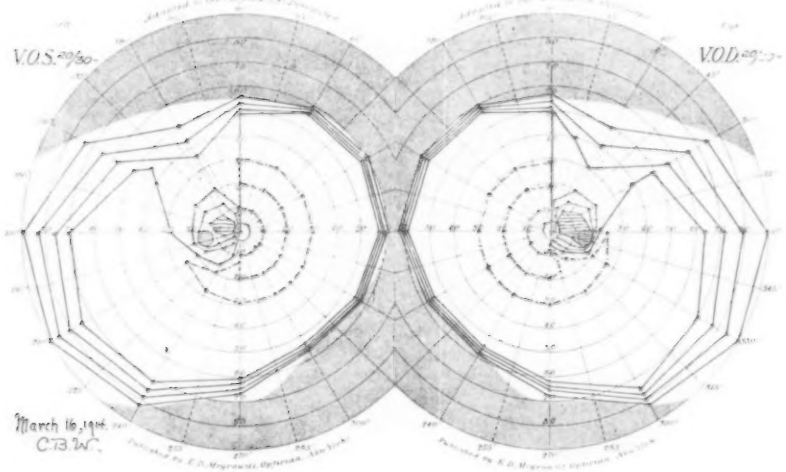

FIG. 19.—Case 4. Fields of March 16, 1914, eleven days after operation, showing fusion of form peripheries to discs of 1 sq. cm. and over, a relative paracentral scotoma for each of them remaining. Dotted lines connect the separate scotomata with the periphery for corresponding disc.

is to the normal) test object, they had fused to the larger discs in the series. In a patient at this stage, therefore, casually examined by the usual 1 cm. disc so often employed in perimetry, the outlines might easily have been regarded as normal, and as likely as not the relative paracentral scotomata shown to be present for the serial discs would have been overlooked. It is clear, of course, that the larger the disc the smaller will be the recorded size of the scotoma. Unfortunately no opportunity for subsequent observations in this case was afforded, and it is quite possible that completely normal outlines were attained.

Cases admitted in Stage IV.—We now come to Stage IV, representing the text-book stage of hemianopsia. Here apparently in many cases the condition lingers, and to this may possibly be attributed the fact that it has so long been regarded as the typical stage of the process. At this stage, moreover, one begins to have some misgivings in regard to restoration, a matter which doubtless depends on the duration of the pressure. In one patient who was seen by one of us five years ago in an advanced Stage III and who declined operation [6], the condition has only just advanced beyond Stage IV, but with so great a lowering of acuity that he eagerly sought the operation which had formerly been advised. This has recently been performed, but despite the thorough evacuation from the lower sella of its soft struma, there has been no immediate improvement in vision such as would have been expected after a similar operation performed earlier in the disease.

Provided, therefore, that the pressure has not been of too long standing, patients in this stage of hemianopsia may slowly improve, as exemplified by Case 1 recorded above, and occasionally the improvement may be very rapid; in some exceptional cases, indeed, normal outlines may be plotted a few days after an operation. This has happened twice in the series, both cases being those in which a cyst was encountered and drained and in which the hemianopsia had been of brief duration—a matter of months rather than of years.

The following case, though belonging in the amaurotic group owing to the left blindness, is one in which the seeing eye, were reliance placed solely on the usual normal disc, would be regarded as showing a typical hemianopsia (barring the mid-temporal field patch, to which further reference will be made). The serial discs, however, not only show that the condition is short of Stage IV, but also how it is that with returning vision the isolated island becomes incorporated in the widening field plotted with the normal ($\frac{1}{4}$ sq. cm. or 0·5 cm.) disc which had revealed the island.

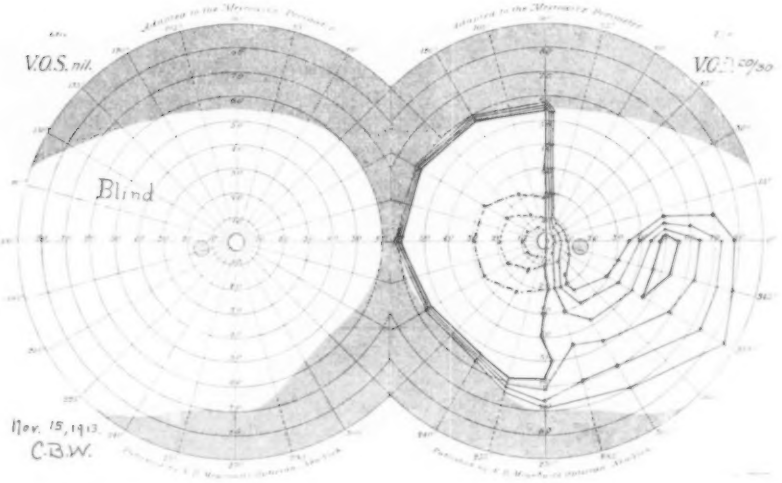

FIG. 20.—Case 5. Fields of November 15, 1913, five days before operation. O.S., Stage VIII; O.D., just short of Stage IV. Note island of retained vision to normal ¼ sq. cm. disc.

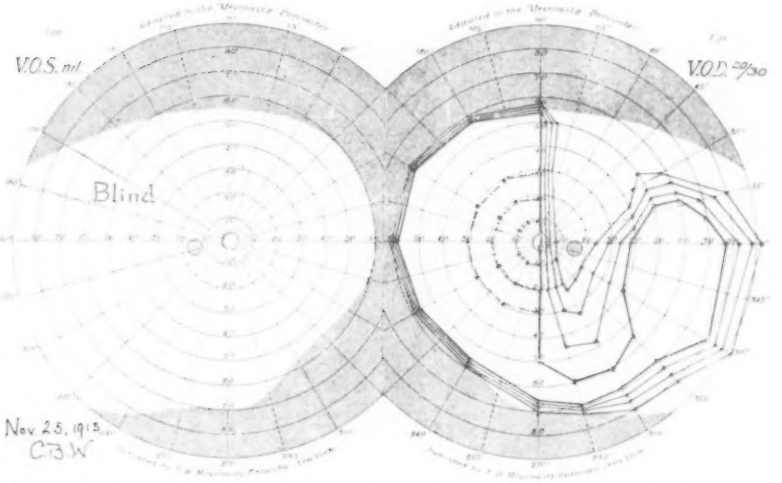

FIG. 21.—Case 5. Fields of November 25, 1913, five days after operation, showing incorporation of former island in lower nasal outshoot from field to ¼ sq. cm. disc.

Case 5 (P.B.B.H., Surg. No. 578).—*Hypopituitarism with primary optic atrophy. Left blindness; right eye short of Stage IV. Operation with improvement.*

November 13, 1913.—Admission of William S. M., aged 36, a mechanical draughtsman, referred by Dr. S. J. Wood, of Cape Town, South Africa, through Drs. James T. Taylor and J. Herbert Fisher,[1] of London. A typical example of adult adiposo-genital dystrophy with headaches, sellar distension, polyuria, asthenia, hypotrichosis, &c.

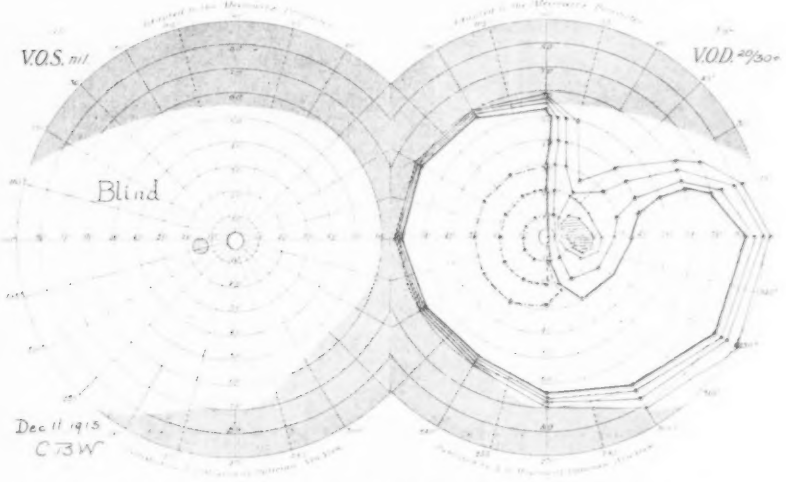

FIG. 22.—Case 5. Fields of December 11, 1913, three weeks after operation, to show recession to an advanced Stage II. Scotomata to the 2 and 4 sq. cm. discs.

Vision began to fail in the left eye about February, 1911, advancing through a temporal hemianopsia to blindness in one year. The right eye was first subjectively affected in June, 1912, seventeen months ago, and has progressed to its present condition.

Examination of eyes.—Pupils wide. *Fundus:* Discs pale, atrophic, moderately pigmented, 7 to 8 D. myopia. *Fields* (fig. 20): No vision on the left; right shows, to the normal $\frac{1}{4}$ sq. cm. disc, a complete hemianopsia with vertical meridian and a retained temporal patch in addition. This patch is concentrically enveloped in the three successive outlines plotted with larger discs. No relative central scotoma. V.O.S. nil, V.O.D. (corrected) $\frac{2}{30}$.

November 20, 1913.—*Operation.* Usual transphenoidal procedure with evacuation of large amount of soft chromophobe struma. No complications.

[1] Mr. Fisher (14) has already recorded the earlier progress of this case when there was still nasal vision in the left eye and the right had normal fields.

November 25.—*Fields* (fig. 21): There is widening of the lower temporal areas and the island has become incorporated in the offshoot from the lower nasal field.

December 2.—Further widening of fields demonstrated.

December 11.—*Fields* (fig. 22) on this date in fairly typical Stage III. V.O.D. (corrected) $\frac{20}{50}$ +. Patient discharged. No subsequent fields possible. Writes of further improvement.

The following case, though it also belongs in the amaurotic group owing to the Stage VIII condition of the left eye, will serve as another illustration of a condition similar to the above though slightly more advanced, for the island here is completely isolated even for large discs.

Case 6 (P.B.B.H., Surg. No. 24).—*Hypopituitarism* (*Which type*) *with optic atrophy. Left blindness; right eye in Stage IV. Operation. Restoration to Stage VII left, and normal right.*

February 25, 1913.—Admission on the recommendation of Dr. F. O. Nagle, of Philadelphia, of R. E. S., aged 23, a recent graduate in medicine. A typical example of adiposo-genital dystrophy (typus femininus). Three years before, during his medical course, his vision began to fail. He brings a series of perimetric charts taken at intervals since that time. Though too roughly plotted to be of value for our purposes they nevertheless show sufficiently clearly a bilateral advance from upper temporal defects to a temporal hemianopsia on the right and to complete blindness on the left, where vision was completely lost by February of 1912. Specific treatment, neosalvarsan, &c., without avail.

Examination of eyes.—No exophthalmos, diplopia, nystagmus, or extrinsic muscular disturbance. *Pupils* slightly dilated; left larger than right. Direct pupillary reaction present on the right, absent on left; indirect reaction present on left, absent on right; hemiopic reaction (rotary shutter) present on right. Refixation (Wilbrand test by use of special electric lights) retarded. Pseudo-refixation present. *Fundus:* O.U., optic disc wide, pale, and sharply outlined. Optic cup wide and shallow. Lamina cribrosa easily made out, but not as sharp as in a toxic primary atrophy. Faint layer of new tissue on disc but no perivascular streaking. Fundus otherwise in good condition. *Fields* (fig. 23): No vision on the left. On the right a hemianopsia with central scotoma, typical in all respects of Stage IV except for the small patch of vision on the horizontal meridian in the midtemporal field. V.O.D. $\frac{20}{200}$.

March 3.—*Operation.* Typical transphenoidal procedure with extensive removal of soft chromophobe struma. Uneventful recovery, with immediate (on recovery from anæsthetic) subjective improvement in vision.

March 6 (third day).—*Field* (fig. 24) on the right has greatly widened. A tongue for the normal $\frac{1}{4}$ sq. cm. disc has shot out from the lower side and

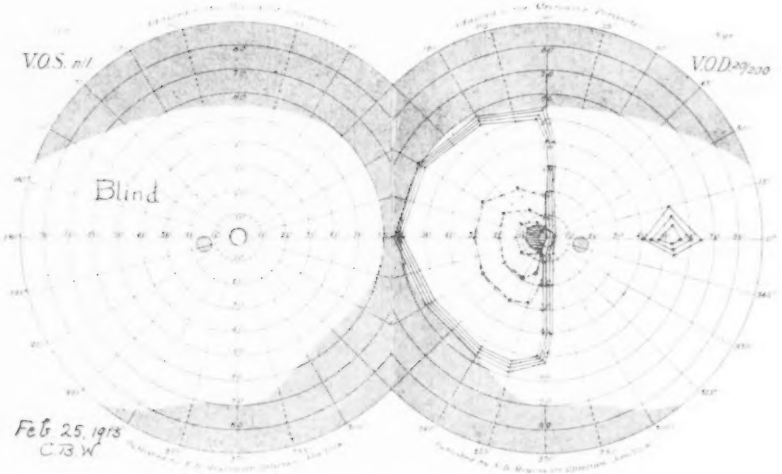

FIG. 23.—Case 6. Fields of February 25, 1913, showing condition before operation. O.S., Stage VIII; O.D., Stage IV. Note retained island in right temporal field.

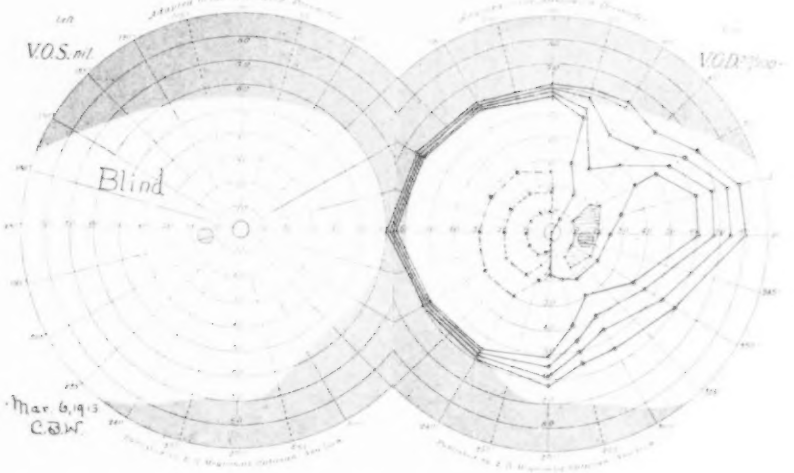

FIG. 24.—Case 6. Fields of March 6, 1913. Three days after operation, showing early great improvement. Note development by widening fields of isolated patch shown in fig. 23.

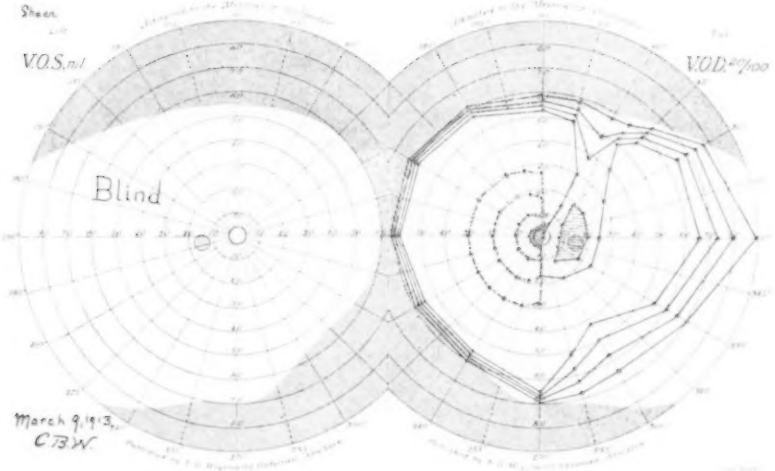

FIG. 25.—Case 6. Fields of March 9, 1913, six days after operation. Paracentral scotoma to 1 sq. cm. disc persists. Note approaching fusion of peripheries to ¼ sq. cm. disc.

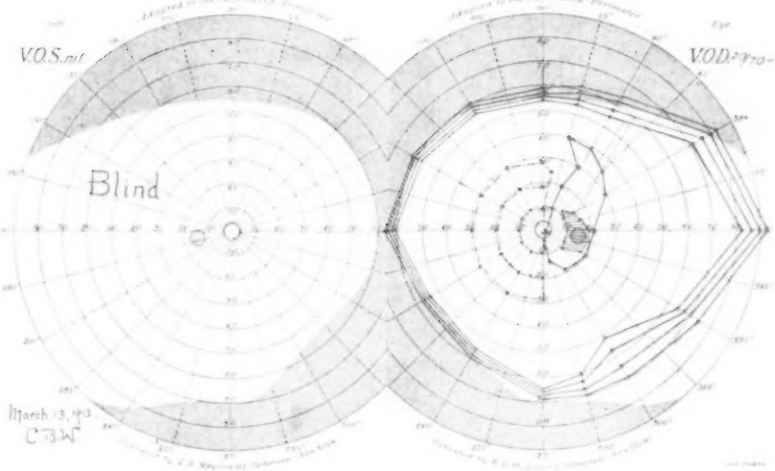

FIG. 26.—Case 6. Fields of March 13, 1913, ten days after operation, showing peripheral fusion of the field to the normal ¼ sq. cm. disc, leaving a relative paracentral scotoma for this as well as for the 1 sq. cm. disc.

enveloped the patch of preserved vision mentioned above. To larger discs (1 sq. cm. and over) the peripheral field, advancing from the sides, has fused around the upper temporal quadrant enclosing a scotoma for the 1 sq. cm. disc. V.O.D. $\frac{20}{1000}$.

March 9 (sixth day).—*Fields* (fig. 25): A still further improvement shown, though fusion not quite complete as yet to the normal $\frac{1}{4}$ sq. cm. disc. V.O.D. remains at $\frac{20}{1000}$, owing to the central scotoma.

March 13.—The peripheral field (fig. 26) has fused even to the smallest disc, leaving relative paracentral scotomata to the two smaller ($\frac{1}{4}$ and 1 sq. cm.) discs. V.O.D. $\frac{20}{70}-$. *Discharged.*

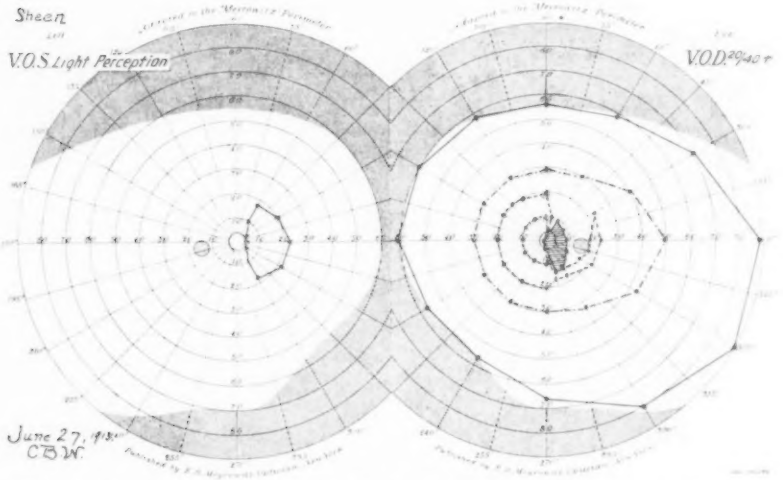

FIG. 27. Case 6. Fields of June 27, 1913, eight weeks after operation, showing normal conditions (apart from the scotoma and the red and green peripheries) largely regained on the right, and return of vision (Stage VII) on the left.

April 26, 1913.—*Reports for examination. Fields* show further improvement with an unexpected return of the pupillary reaction and of a nasal patch of vision on the left, where blindness had been complete since November of 1912. On the right the peripheries are fairly normal, with a persisting enfeeblement of central vision and a relative paracentral scotoma to both $\frac{1}{4}$ and 1 sq. cm. discs. V.O.D. $\frac{20}{70}-$.

June 27, 1913.—*Reports for examination. Fields* (fig. 27) improved over last note. The patch of vision on the left persists unchanged. On the right the form periphery is normal and also the blue periphery to $\frac{1}{4}$ sq. cm. discs. The red field projects on to the lower temporal field and there is only half vision for green. There is also a small relative scotoma for the $\frac{1}{4}$ sq. cm. disc. V.O.S., light perception, V.O.D. $\frac{20}{40}+$.

Subsequently, though there was some further progress it was very slow, with but little variation in the later fields. However, by October the acuity had risen to $\frac{20}{40}$.

The fields of *October* 16, 1913, show a persistence of the scotoma, accounting for the slightly lowered vision in what would otherwise be a normal field and eye. This observation represented the high tide of improvement, and the condition held there for the following nine months. At the last examination, *August* 4, 1914, a slight lowering of the acuity was noted, with a measurable increase in size of the scotoma, but otherwise the fields had remained as in June of 1913.

Comment.—As in the preceding case, here again an interesting feature of the blind temporal field is the patch of retained vision on the horizontal meridian seen on the pre-operative charts; and the manner in which this small area is engulfed by the upward swing of the tongue of vision in the process of recession to Stage III is very clear in the post-operative series. Doubtless had an observation been made on the first or second day after the operation, the transition would have been still more apparent.

In the following case the manner in which such a patch becomes incorporated in the widening field is particularly clear. It shows, moreover, not only that these patches may be bilaterally represented, but also that in the process of restoration such an isolated patch may chance to be recorded if the examination happens to coincide with its appearance. Unfortunately we were only able to observe this patient for less than three weeks, and we are unaware of the later changes.

Case 7 (Corey Hill Hospital).—*Hypophysial struma with optic atrophy. Fairly symmetrical bitemporal defect short of Stage IV. Operation. Improvement.*

December 17, 1912.—Admission of G. E. H., aged 32, a business man, referred by Dr. C. L. Chambers, of Detroit. There were very few glandular manifestations in the case, the greatly distended sella and the bitemporal hemianopsia being the chief distinguishing features.

He had had "trouble with his eyes" for six or seven years, and had consulted numerous specialists, who had changed his glasses. There had been occasional periods of diplopia.

Examination of eyes.—Pupils equal and reactions normal. *Fundus* shows considerable pallor of nerves. *Fields* (fig. 28) show a fairly symmetrical bitemporal defect approaching Stage IV, with an island of vision for the largest disc alone on the right temporal field, and also a central scotoma. V.O.S. $\frac{20}{40}$, V.O.D. $\frac{20}{70}$.

January 18, 1913.—*Operation.* Sellar decompression without removal of struma. Uneventful recovery.

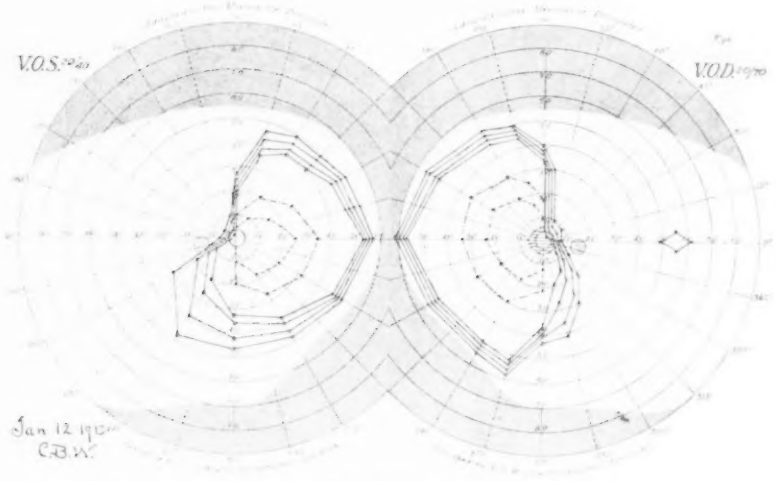

FIG. 28.—Case 7. Fields of January 12, 1913, before operation. Both eyes just short of Stage IV. Note island of vision in right temporal field.

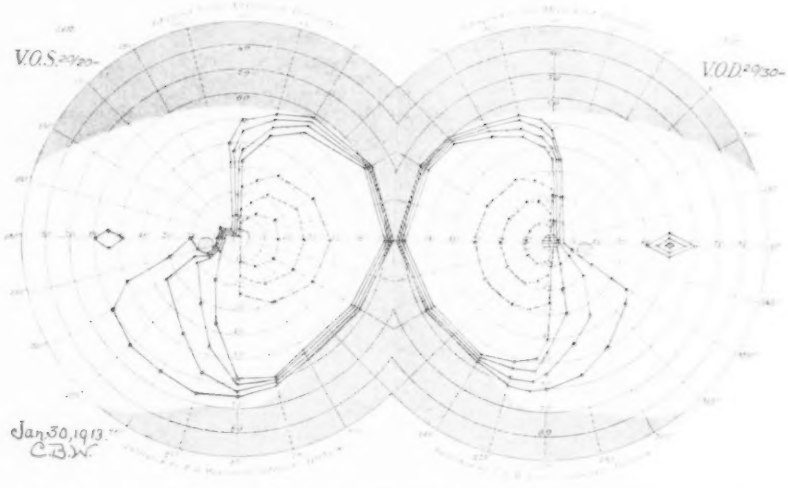

FIG. 29.—Case 7. Fields of January 30, 1913, twelve days after operation, showing widening of the lower temporal peripheries and the appearance of a symmetrical island on the left.

January 26.—*Fields* show some widening of the peripheries, and the acuity has considerably improved. V.O.S. $\frac{20}{50}$, V.O.D. $\frac{20}{50}$.

January 30.—*Fields* (fig. 29) show further widening and the appearance of an island for the largest disc alone in the left temporal field, while the island on the right has regained vision for smaller discs. Acuity unchanged. Patient discharged.

February 5.—*Reports for examination. Fields* (fig. 30) show further change with approach to Stage III and incorporation of the island on the right in the widening field for the largest disc. V.O.S. $\frac{20}{15}$, V.O.D. $\frac{20}{20}$ +.

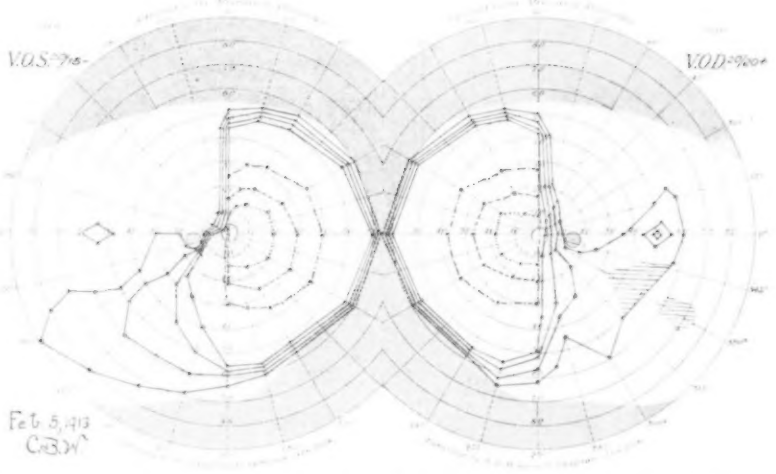

FIG. 30.—Case 7. Fields of February 5, 1913, eighteen days after operation, showing further recession toward Stage III, with engulfing of island on the right by the 16 sq. cm. disc.

Comment.—That these patients do not all do as well after operation as the foregoing seven cases might seem to indicate is shown by the following example in which the condition advanced despite the two operations which were undertaken. The case has been chosen to illustrate Stages V and VI of the process, and though it has been taken from the amaurotic group, the history shows that a bitemporal defect has once been observed. Explanations for the failure of improvement probably lie in the long duration of the process and in the presumable suprasellar extension of the struma.

Case 8 (P.B.B.H., Surg. No. 37).—*Hypopituitarism. Chromophobe struma with probable suprasellar extension. Optic atrophy. Stage V advancing to Stage II despite operation.*

October 16, 1912.—Admission of J. D. A., aged 52, a salesman, recommended by Dr. C. M. Carlow, of Minneapolis.

An example of advanced adult adiposo-genital dystrophy with hypotrichosis, obesity, impotence, somnolence, psychoses, complete loss of sellar outlines, &c.

Subjective loss of vision was first observed in the fall of 1910, a perimetric examination at the time showing left hemianopsia: right eye normal. By December of 1911 vision began to fail on the right, and in June of 1912 a bitemporal hemianopsia was present (Dr. Casey A. Wood). The condition progressed, and three weeks before admission vision had completely faded on the right, so that though the process began on the left, the right eye finally outstripped the left.

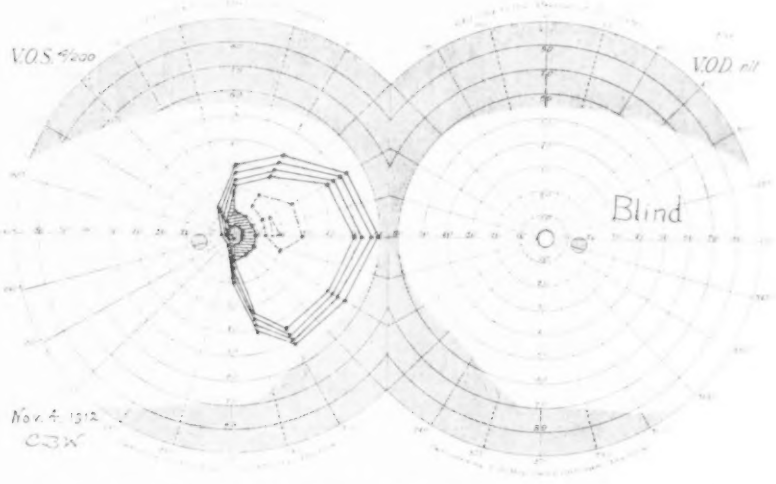

FIG. 31.—Case 8. Fields of November 4, 1912, showing the Stage V condition at the first admission unaltered by operation.

Examination of eyes.—No exophthalmos; movements free and complete; no nystagmus. O.D. turns in slightly. Right pupil dilated and inactive to direct light, but active consensually with O.S. Pupil O.S. smaller, and gives good direct, but no consensual reaction. *Fundus.* Extreme pallor. Lamina cribrosa hazily seen. Typical "primary optic atrophy." *Fields* (fig. 31) show an advanced Stage V on the left; colours in a small area apparent only to large discs. V.O.S. $\frac{3}{200}$, V.O.D. nil.

October 21, 1912.—*Operation.* Usual transphenoidal procedure with extirpation of large amount of soft chromophobe struma. Uncomplicated recovery. On the morning after operation some light perception in the blind eye, but

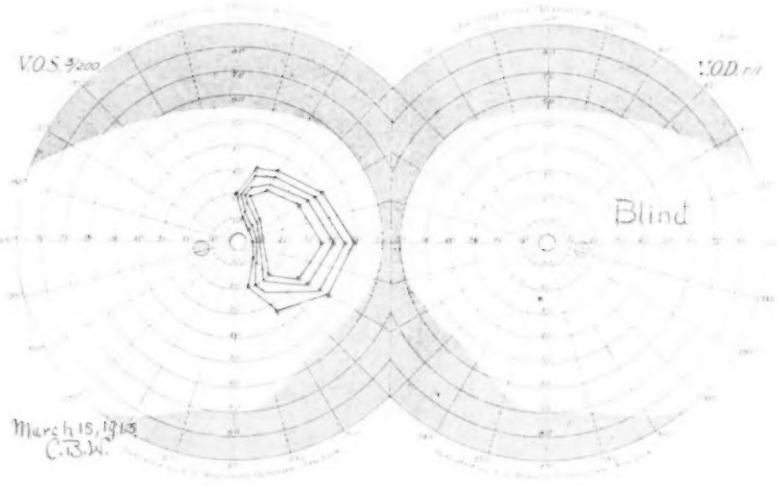

FIG. 32.—Case 8. Fields on second admission, March 15, 1913, showing advance to Stage VI.

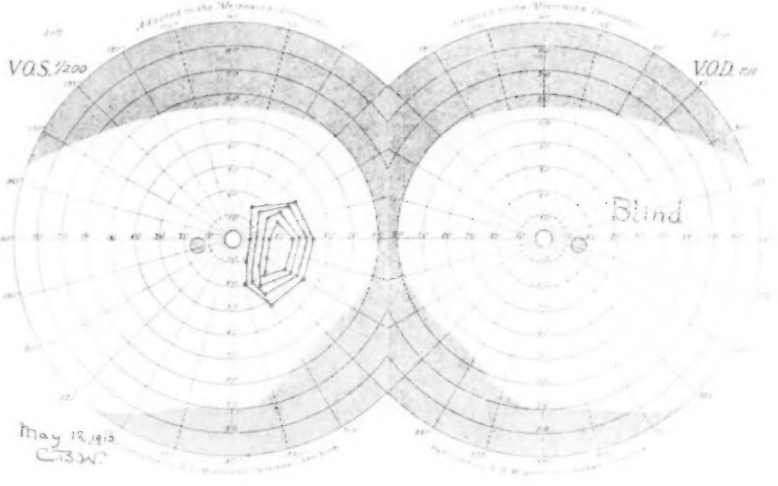

FIG. 33.—Case 8. Fields of May 12, 1913, after second operation, showing advance to Stage VII.

this was soon lost. Fields of *October* 26 and *November* 4 show a slight widening of the peripheries, but no significant change. Discharged.

March 15, 1913.—*Readmitted.* His general condition, despite pituitary feeding, has progressed unfavourably. The vision has remained almost stationary until the past few weeks, since when it has failed rapidly. The *field* has considerably shrunken, approaching Stage VI (fig. 32). V.O.S. $\frac{3}{200}$.

April 14, 1913.—*Second operation.* Transphenoidal route re-entered, with further extensive removal of intrasellar struma. Uneventful recovery, but progressive constriction of fields (fig. 33) and a further lowering of vision to V.O.S. $\frac{1}{200}$ at the time of discharge, *May* 12, 1913.

September 2, 1913.—By letter. Mere light perception. Symptomatic evidences of intracranial involvment.

Comment.—The above case is given largely to emphasize the need of early operation in the presence of a greatly enlarged sella, for this condition represents a comparatively simple surgical problem. With delay an intracranial invasion by the œdematous struma is likely to occur, and there is then little hope for preservation of vision, or, indeed, for a long preservation of life. However, under favourable circumstances, even an advance to Stage VII may not be incompatible with improvement, as the succeeding history will show. It illustrates a rapid recession after operation from Stage VII to Stage III—an unusual degree of improvement in our experience after such an extensive involvement of the nerves has become apparent.

Case 9 (P.B.B.H., Surg. No. 1A).—*Hypopituitarism with chromophobe struma, optic atrophy, adiposity, &c. Operation. Recession from Stage VII to Stage III.*

January 1, 1913.—Admission of A. S., aged 61, proprietor of a restaurant, recommended by Dr. F. W. Marlow, of Syracuse.

A typical example of hypophysial adiposity with advancing blindness and a widely distended, largely absorbed sella. Failing lateral and central vision had been observed for one year. A month before admission the fields (Dr. Marlow) showed a fairly symmetrical bitemporal defect in Stage IV.

Examination of eyes.—No exophthalmos, von Græfe or strabismus. Slight nystagmus. Pupils react to light, directly and indirectly, consensually best from right to left. Direct pupillary reaction best on right. *Fundi:* O.D., disc slightly injected but lamina cribrosa sharp in a pale shallow optic cup of moderate width. Vascular condition good: macular region and peripheral fundus clear. O.S., disc very pale, margins sharp; lamina cribrosa clearly outlined in a shallow, wide, glistening optic cup. Fundus otherwise as in O.D. *Fields* (fig. 34) show a condition on the left approaching Stage VII and on the right a typical though advanced Stage IV, with considerable macular involvement. V.O.S. $\frac{3}{200}$, V.O.D. $\frac{20}{200}$.

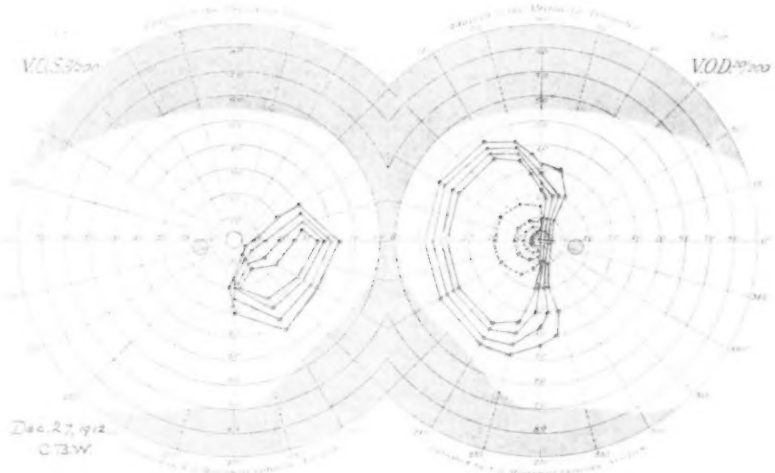

FIG. 34.—Case 9. Fields of December 27, 1912, before operation. O.S. approximating Stage VII; O.D. in an advanced Stage IV.

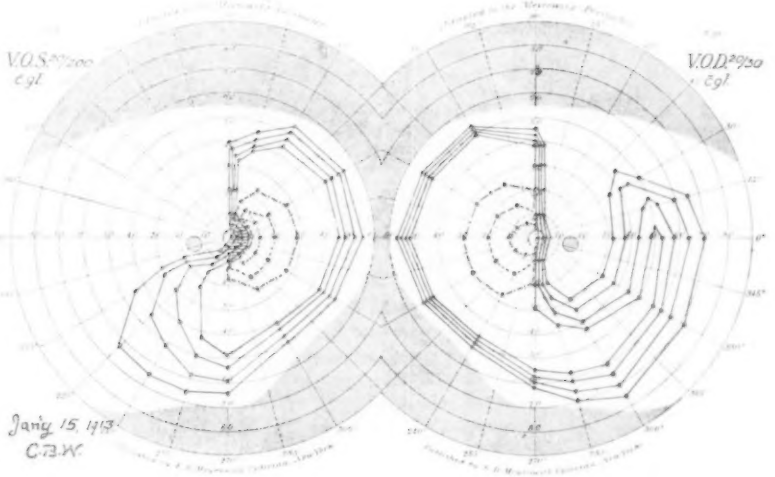

FIG. 35.—Case 9. Fields of January 15, 1913, four days after operation, showing recession to a full Stage III on the right, and on the left an astonishing change from Stage VII to one just short of Stage III.

January 11, 1913.—*Operation.* Usual transphenoidal procedure with partial extirpation of chromophobe struma. Uneventful recovery.

January 15 (Fourth day).—*Fields* show an astonishing improvement (fig. 35), particularly in the left eye, which has receded from Stage VII to an advanced Stage III. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{20}{50}$.

January 19 (Eighth day).—*Fields* (fig. 36) and vision show further improvement. V.O.S. $\frac{20}{70}$, V.O.D. $\frac{20}{20}$.

January 23. (Twelfth day).—*Fields* (fig. 37) show an advance to Stage III on the left with increase in acuity from $\frac{20}{200}$ to $\frac{20}{70}$ in twelve days; on the right the form periphery has fused to the largest disc, and the vision has increased from $\frac{20}{200}$ to $\frac{20}{20}$ in the twelve days.

Patient discharged. No further report or reply to inquiry.

Comment.—It is regrettable that this patient has been lost sight of, for it would have been interesting to see whether a still further recession, possibly to Stage I or Stage II, did not occur in the left eye. It is presumable that nearly normal conditions may for a time have been regained in the right eye, where so far as acuity, outlines and duration of the process were concerned, the condition corresponded closely with that of the right eye in Case 6 already recorded and which fortunately could be followed through.

The following case report serves to show that even when Stage VIII has been reached, with blindness, one may still cling to the hope that a certain degree of useful vision may be restored, provided the implication of the nerves has not been of too long duration, and provided that a favourable condition such as a cyst is encountered.

Case 10 (J.H.H., Surg. No. 28,382).—*Cystic struma with hypopituitarism. Pronounced neighbourhood symptoms. Operation. Partial restoration of vision after blindness.*

September 13, 1911.—Admission of Mrs. E. J. W., aged 44, referred by Dr. D. B. Smith, of Setart, West Va. A characteristic case of hypopituitarism with adiposity, amenorrhœa, somnolence, &c. Complete absence of sellar outlines. Severe headaches began in 1909, but ceased after a year, when her vision began to fail, according to the history advancing as a bitemporal blindness. Since December of 1910 vision has been practically *nil*, though until three weeks before entrance she could distinguish moving objects. She states that vision faded first in the left eye and then in the right.

Examination of eyes.—Movements complete but slight exophoria. No nystagmus, ptosis or exophthalmos. Some divergence of globes. Pupils equal and moderately dilated. Right pupil reacts feebly to light, and there is bare perception of strong light; left pupil has sluggish indirect reaction. *Fundus:* O.U., optic disc pearly white; margins irregular but sharp and pigmented; lamina cribrosa sharply outlined; no œdema of the nerve-head.

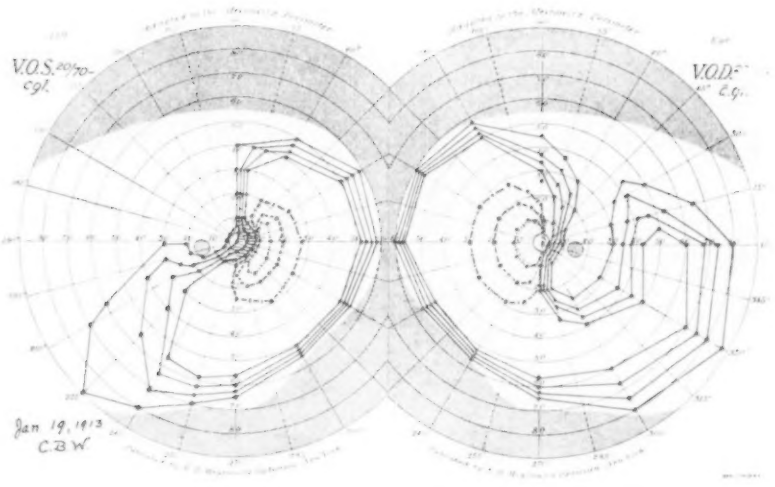

FIG. 36.—Case 9. Fields of January 19, 1913, eight days after operation, with further improvement.

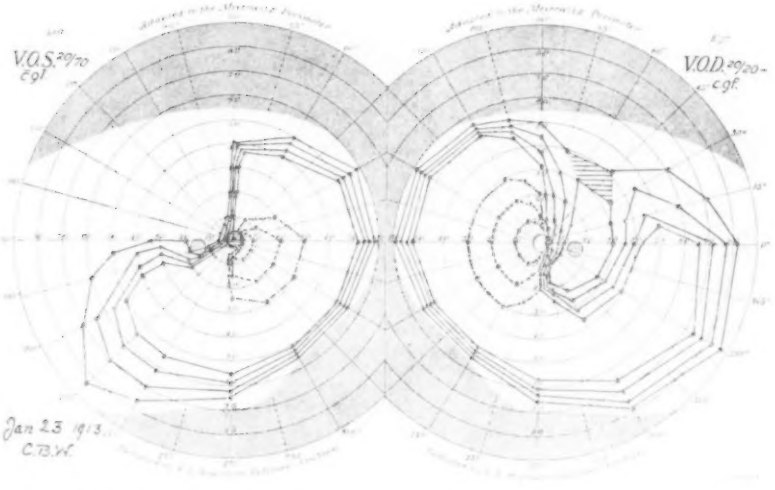

FIG. 37.—Case 9. Fields of January 23, 1913, twelve days after operation. Showing O.S., an advance to Stage III; O.D., a fusion of the peripheries of the larger disc.

Vessels and peripheral fundus show no change. *Fields:* Perimetry precluded. Faint light perception, limited to right temporal retina.

September 18.—Owing to a misinterpretation of the case and thinking it one of frontal lobe tumour, Dr. Sharpe performed a right exploratory osteoplastic craniotomy with a decompression. Her condition remained unaltered.

October 11.—*Operation.* Evacuation of cystic struma by transphenoidal approach. Patient could count fingers the afternoon of operation. No post-operative complications. Subjective improvement in vision continued. Perimetry was first employed on October 17 1911.

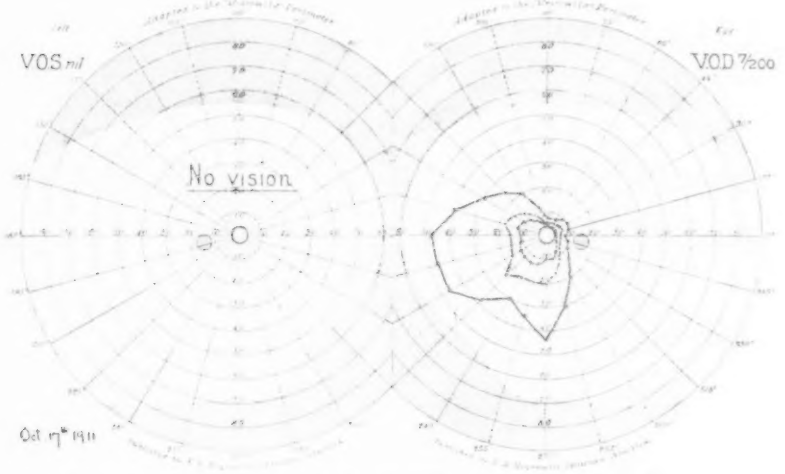

FIG. 38.—Case 10. Fields of October 17, 1911, with 0·5 cm. discs six days after operation. Pre-operative vision *nil*.

October 17.—*Fields* (fig. 38) show the restoration of something more than a quadrant of lower nasal vision with the macula included in the field in the right eye. V.O.S. *nil*, V.O.D. $\frac{7}{200}$.

October 30.—*Fields.* No change in outline. V.O.D. $\frac{15}{200}$.

November 6 and *November* 16.—*Fields* show no change. V.O.D. $\frac{15}{200}$. Discharged.

March 6, 1912.—By letter. Vision remains the same. General condition much improved.

Comment.—In this patient the condition in the least affected eye, the one in which vision returned, had advanced to a point at which shadows were no longer perceptible and fields could not be charted, in fact almost to Stage VIII.

It is one of the older cases and unquestionably from what we now know more careful perimetry would have shown a relative central scotoma, and it is possible too that fixation was not accurate and that the plotted area should have been registered farther to the nasal side.

There are several other cases in the series in which a modicum of vision has slowly returned in an absolutely blind eye. Indeed in one of the patients in the homonymous group, a woman who was completely blind before operation, some vision returned in both eyes. In only one other case in the series, in addition to this Case 10, was the return of vision after blindness accompanied by a return of colour perception as well as of perception for form.[1]

When, in these blind patients, despite the widened pupil some reaction to light is still retained the prognosis for some restoration of vision is naturally better than when the pupillary reflex is lost, for it implies that there are still some functionally intact fibres. We have seen, however, by the return of a patch of vision in the left eye in Case 5, which had been blind for a year before the operation, and in which the direct light reflex was completely abolished, that some light perception may be regained even under these more unpromising conditions.

Bizarre types of field distortion.—The foregoing ten cases have been selected as fairly representative, at the time of admission, of the stages into which we have divided hemianopsia. Attention may again be called to the fact that they all represent examples of pituitary disease (acromegaly, or its reverse clinical state) in which the sella has been widely distended by a tumefaction or so-called struma of the gland itself. Such a lesion in the course of its enlargement naturally presses upon the chiasm from below and behind, affecting the various fasciculi of the nerve in fairly definite progression.

It must be realized, however, as has been already emphasized, that when the gland is subject to a marked enlargement or tumefaction it is capable of bursting through the dural capsule with ultimate extensive invasion of the intracranial chamber. Even under these circumstances the nerves may entirely escape from functional pressure implication, for they are capable of extreme anatomical distortion without demonstrable perimetric change. Still, when the growth has extended through the dural envelope and made its way upward it may envelop

[1] This other case was recorded as Case 22 in "The Pituitary Body and its Disorders," 1911. Blindness had been complete for only two days, and there was sufficient return of vision in each eye to permit of perimetry.

the chiasm and its four branches in irregular ways and produce bizarre types of field deformation which deviate from the regulation stages which we have heretofore described.

In the following case, for example, the dorsal uncrossed fasciculi became involved before the ventral fasciculi, leaving a fairly clear-cut superior nasal quadrantal field of preserved vision, and the tendency toward restoration appeared in the upper nasal field rather than in the lower, as is the rule.

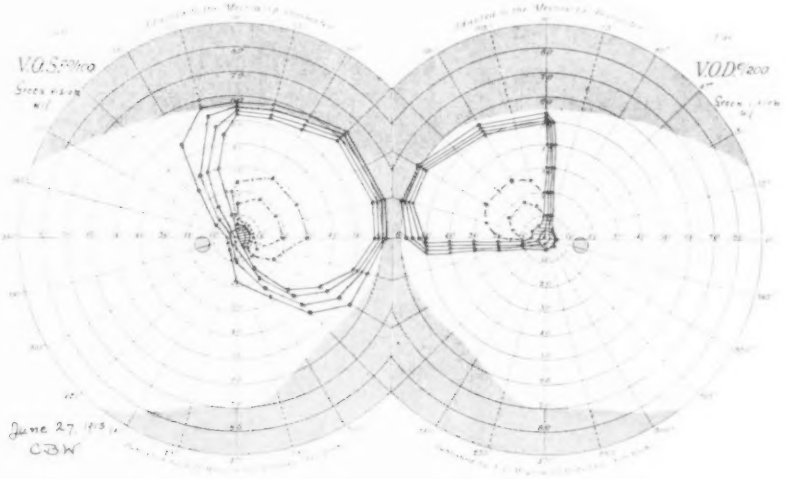

FIG. 39.—Case 11. Fields of June 27, 1913, three weeks before operation, showing unusual though fairly symmetrical type of bitemporal defect.

Case 11 (P.B.B.H., Surg. No. 203).—*Struma with hypopituitarism and unusual field deformation.*

June 27, 1913.—Admission of Morris B., aged 43, a storekeeper, referred by Drs. W. Simon and Charles H. May, of New York. A fairly typical example of hypophysial insufficiency, with drowsiness, headaches and loss of vision as the chief complaint. Lowering of vision first noticed three years ago, with progressive advance. Diplopia, present for one year, has now disappeared. A greatly enlarged sella with absorbed dorsum.

Examination of eyes.—*Pupils* small, equal: sluggish reactions. Hemiopic reaction in both eyes, best seen on the left. No apparent oculomotor palsies. *Fundus:* marked pallor of discs with crescentric pigment deposit: veins full and tortuous. Optic cup filled with new tissue: lamina cribrosa obscured. No œdema or measurable swelling. *Fields* (fig. 39) show a fairly symmetrical

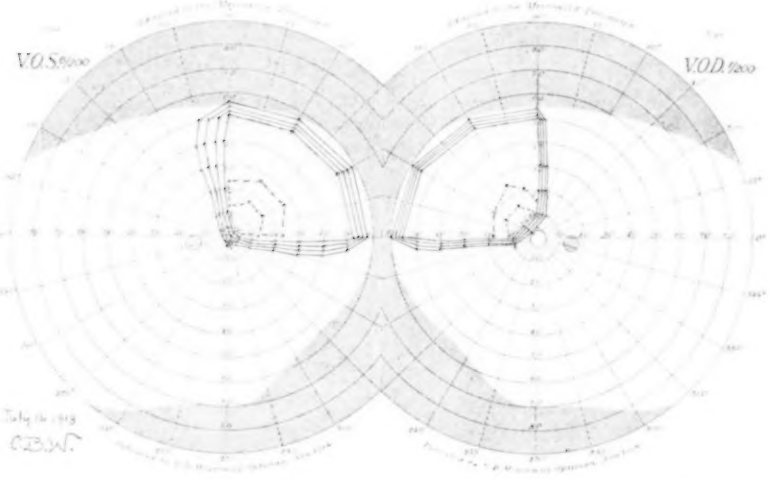

FIG. 40.—Case 11. Fields of July 16, 1913, three days before operation, showing further advance in untreated lesion.

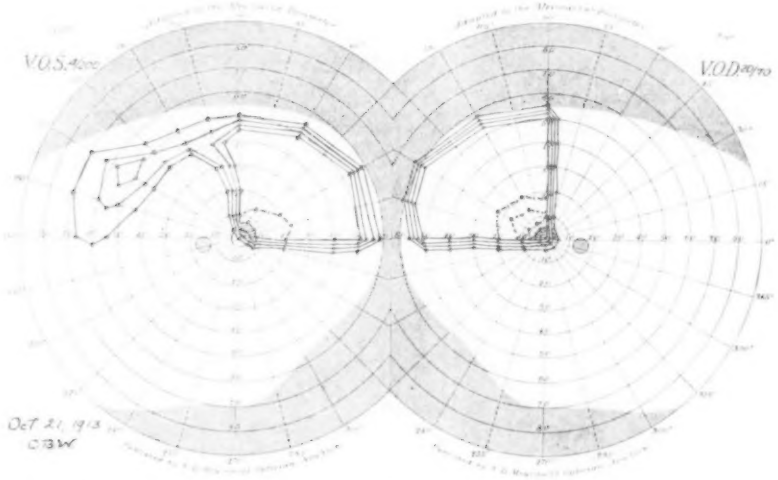

FIG. 41.—Case 11. Fields of October 21, 1913, showing O.S. tendency toward upper temporal field restoration.

preservation of vision in the upper nasal quadrants, with enfeeblement of the macular bundle on each side. V.O.S. $\frac{20}{100}$, V.O.D. $\frac{6}{200}$.

Operation advised and deferred.

July 15.—*Readmission.* Condition advanced. *Fields* (fig. 40) now limited to the quadrants and show more extensive macular impairment. V.O.S. $\frac{6}{200}$, V.O.D. $\frac{1}{200}$.

July 19.—*Operation.* Usual transphenoidal procedure with partial extirpation of characteristic chromophobe struma. Uneventful convalescence.

July 24.—*Fields* unchanged. Some subjective improvement in vision.

August 2.—*Fields* unchanged. Improvement in vision on the right. V.O.S. $\frac{4}{200}$, V.O.D. $\frac{15}{200}$. *Discharged.*

October 21.—*Reports for examination. Fields* (fig. 41) to larger discs have shot out over left upper temporal quadrant, but there is no central visual improvement in this eye, unlike the other, where the outline remains unaffected. V.O.S. $\frac{4}{200}$, V.O.D. $\frac{20}{70}$.

Comment.—The secondary changes in the fundi, the presence of diplopia, as well as the unusual field defects, make it evident that the lesion had extended upward and around the nerves. Hence our operation did not offer much chance of improvement. The way in which the field peripheries, according to the last post-operative observation, have spread out on to the left temporal field resembles, though upside-down, the manner of restoration which occurs in the standard type of recession towards Stage III.

Many other examples of irregular fields might be cited from this group of primary intrasellar enlargements in which the nerves have become involved by an upward extension of a primarily intrasellar lesion, but this single case may perhaps suffice.

One thing further may deserve a word of mention in connexion with these cases, and that is the not infrequent combination of a normal with a blind eye, of which condition there are three or four illustrative cases in the series, not to mention a few others in which the field of the seeing eye has begun to show an early slant (Stage I) of the upper temporal periphery. It is difficult, of course, in these cases, without a definite history of the way in which the field of the blind eye first became involved, and without waiting for changes in the seeing eye, to tell whether the case really belongs in the homonymous or bitemporal group. Unless associated with a congenital absence of chiasmal crossing, which is rare, the condition of course must indicate an implication of a single optic nerve anterior to the chiasm, for otherwise the temporal field in the seeing eye would necessarily have shown constriction.

(*b*) *The suprasellar tumours causing bitemporal hemianopsia.*—We come now to a group of cases in which the chiasmal involvement is due to a lesion of quite a different nature—namely, to a superimposed (rather than an intrasellar) tumefaction, the most characteristic example of which, the *Hypophysenenganglumor* of Erdheim, is usually a cystic growth of pharyngeal anlage situated in the interpeduncular space, where it often surmounts a sella which gives fairly normal outlines to the X-ray. On first thought one would assume that in these cases the perimetric defect would first appear in the lower temporal field, but this we have not found to be the case. Indeed, our few most striking examples of defects starting in the lower quadrants have occurred in conjunction with large primarily intrasellar strumas.

In this group of patients with superimposed tumours there occur many illustrations of field defects which correspond in all respects with the typical stages, examples of which have already been given. From a surgical standpoint, however, these cases are far less favourable than the others, and consequently there have been fewer notable instances of post-operative improvement in perimetric conditions. Moreover, as a rule, the condition is a slowly progressive one, without particular discomforts, and therefore is often neglected, so that in many of the patients one or both eyes at the time of admission have advanced well beyond Stage IV. As is true also of the group we have just considered, so in these cases the process by no means advances with bilateral symmetry, for though the stages occur in characteristic sequence one eye is usually well in advance of the other.

One or two examples may be given. The first illustrates a satisfactory ultimate post-operative recession in each eye from Stage IV to approximately Stage I.

Case 12 (J.H.H., Surg. No. 28,986).—*Hypopituitarism with interpeduncular cyst. Operation. Meningitis. Restoration of vision.*

December 26, 1911.—Admission of James A., aged 19, a student, referred by Drs. Walker and Jelliffe, of New York. An example of hypophysial insufficiency of the puerile type, with somewhat enlarged and distorted sella showing a suprasellar shadow. Severe headaches for four or five years, aural pain with tinnitus, &c. The hemianopsia was not detected until the present year, though vision began to fail in 1909, first in the right and then in the left eye. Rapid advance of late.

Examination of eyes.—Pupils equal: normal reactions to light and accommodation; hemianopic pupillary reaction present. *Fundi:* Pallor of both discs, with sharp pigmented margins; considerable new tissue with obscuration of physiological cup and lamina cribrosa: disc elevated 2 D. with

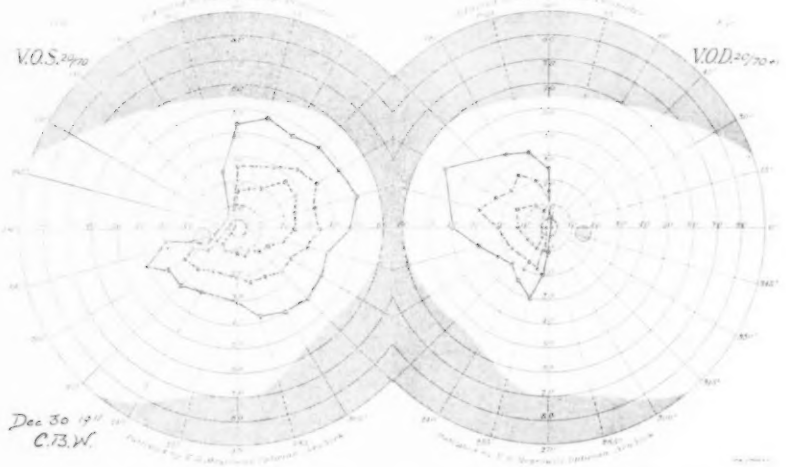

FIG. 42.—Case 12. Fields of December 30, 1911, four days before operation. O.S., Stage III; O.D., Stage IV. Constriction possibly due to associated low grade papilloedema.

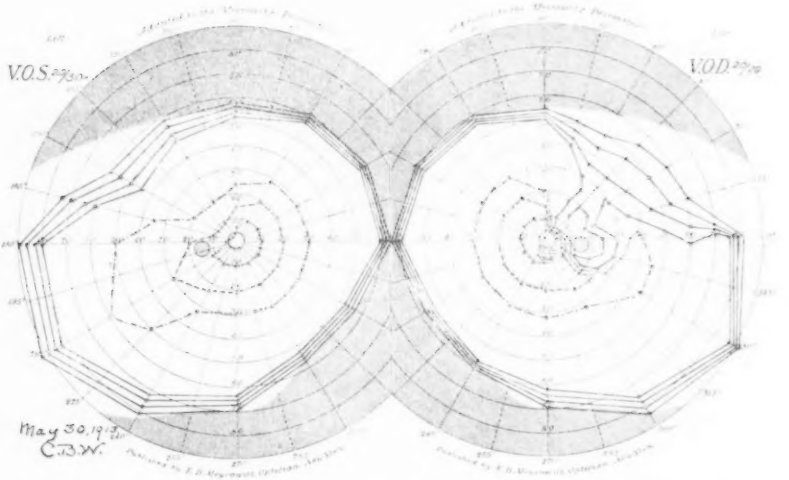

FIG. 43.—Case 12. Fields of May 30, 1913, sixteen months after operation, showing final recession of the process approximately to Stage I.

hyperopic astigmatism. *Fields. December* 26 and 30 (fig. 42) show Stage III approaching Stage IV on the left, and Stage IV on the right. V.O.S. $\frac{20}{70}$, V.O.D. $\frac{20}{70}$ +.

January 4, 1912.—*Operation.* Attempted transphenoidal procedure, abandoned owing to anatomical peculiarities necessitating further X-ray studies.

January 10.—*Second operation.* Re-entry by former route; evacuation of large cyst after cerebrospinal space had been opened. Immediate great improvement in vision, but a cerebrospinal rhinorrhœa became established and after a few days, before perimetry was used, there occurred a sharp attack of cerebrospinal meningitis, from which there was a final recovery.

Fields taken on *January* 31, *February* 3 and 5 (bedside perimetry) and on *February* 15 and 26, show a progressive improvement, particularly in the left eye, the temporal hemianopsia on the right persisting. Patient discharged.

He was subsequently seen and examined on several occasions, no further change in the fields being observed, but as there was some recrudescence of headaches and some continued hyperæmia of the discs on *April* 29 a sub-temporal decompression was performed.

May 30, 1913.—*Reports for examination.* Has been completely free from discomforts since the decompression. His vision has also greatly improved. *Fields* (fig. 43) show a recession to Stage I, the right being somewhat behind the other.

Comment.—The first fields were taken before we came to use our more refined methods, and, on the left, serial discs would probably have shown wider peripheries. Moreover, in view of the lowered vision to $\frac{20}{70}$, there was probably a relative central haziness which our methods of three years ago did not detect or record. Doubtless the further story was considerably complicated by the meningitis which occurred, and unjustifiable risks—then hardly appreciated—were assumed in attacking a lesion of this kind from below. The final recession of the process to about Stage I in both eyes must have taken place slowly after the patient's recovery from the infection and his discharge from the hospital, and we have no records of the intervening stages. It is known that such recessions may take place spontaneously without operation—a matter to be touched upon later.

The following history shows that in cases of this kind the advancing field deformations may not conform to the typical stages which we have described. As in the foregoing, here also the condition was possibly complicated by a superimposed secondary atrophy.

Case 13 (P.B.B.H., Surg. No. 235).—*Hypopituitarism with congenital suprasellar cystic tumour. Transfrontal operation. Fatality.*

July 11, 1913.—Transfer of Dora A., aged 14, from Dr. Christian's medical

service. A small, undeveloped and undersized child, a previous inmate of many hospitals and the victim of many diagnostic errors—"diabetes insipidus," "constipation and obesity," "infantilism," "progressive emaciation," "cerebellar tumour," "tuberculosis," &c.

Formerly a very fat child, she has progressively become emaciated since 1910. For some years there have been headaches with vomiting, polyuria and polydipsia and disturbance of vision. She was brought to the hospital in a semi-conscious condition, from which she slowly emerged as a bright and attractive child. An X-ray shows the shadow of a suprasellar tumour [16] above a pituitary fossa of fairly normal outline.

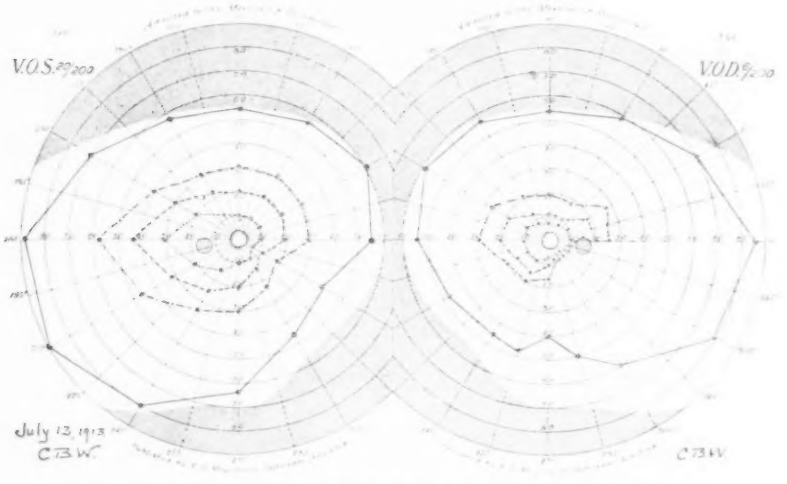

FIG. 44.—Case 13. Fields of July 13, 1913, plotted for $\frac{1}{4}$ sq. cm. disc only. Showing insignificant deformations on the first admission.

Disturbances of vision recognized for several years. Many attempts to correct by glasses. Diagnosis of "low grade optic neuritis" made in May, 1911. No history of diplopia.

Examination of eyes.—Pupils unequal, left larger than right. Reactions normal. Myopic astigmatism. *Fundi:* Discs pale and glistening. Lamina cribrosa obscured, optic cup obliterated, and some new tissue formation along the vessels, which are small. Marginal pigment upheaval. Optic atrophy, suggestive of previous optic neuritis. *Fields* (fig. 44) for normal $\frac{1}{4}$ sq. cm. discs only, show no especial constriction but an unchartable central scotoma on the right. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{6}{200}$ (uncorrected).

July 16.—*Operation.* Right subtemporal decompression revealing a tense and wet brain. Subsequent to this operation there was complete freedom

from the former headaches. The fields of *August* 16 showed no further change. Discharged.

August 22.—*Reports for examination. Fields* (fig. 45), plotted with normal $\frac{1}{4}$ sq. cm. discs only show a marked advance in the constriction with especial implication of macular areas. V.O.S. $\frac{20}{200}$, V.O.D. $\frac{5}{200}$.

September 22.—*Readmission.* Return of pressure symptoms. Decompression area tense. *Fields:* September 29 (fig. 46), plotted for normal $\frac{1}{4}$ sq. cm. discs alone, show further advance. V.O.S. $\frac{10}{200}$, V.O.D. $\frac{1}{200}$ (uncorrected).

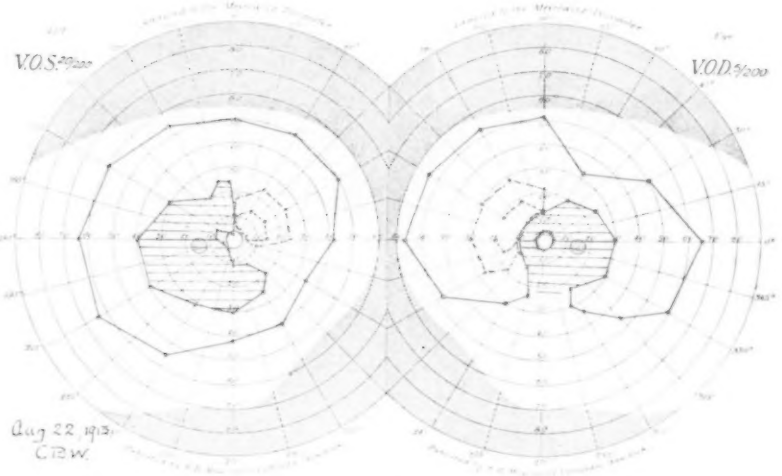

FIG. 45.—Case 13. Fields of August 22, 1913; plotted for $\frac{1}{4}$ sq. cm. discs only. Showing marked advance in five weeks with chief loss in paracentral areas.

October 21.—Periods of intense pressure symptoms have alternated with more comfortable periods, but vision has been failing rapidly. *Fields* (fig. 47) show progressive irregular advance. A small area of preserved central vision permits fixation, so that perimetry is still possible. V.O.S. $\frac{10}{200}$, V.O.D. $\frac{1}{200}$ (uncorrected).[1]

November 4.—Further loss of vision. Fields about as at last note.

[1] Some discrepancies may be observed in the charts for the case due to the fact that perimetry was particularly difficult owing to the child's inattention from drowsiness and asthenia. Thus in the right eye on August 22 (fig. 45), there was in all probability a bridge of vision enclosing the paracentral scotoma, for this was still demonstrable on the test of September 29 (fig. 46). Fortunately up to the last test sufficient macular vision was preserved to make central fixation possible, but the employment of serial discs was precluded owing to the ease with which the child became fatigued.

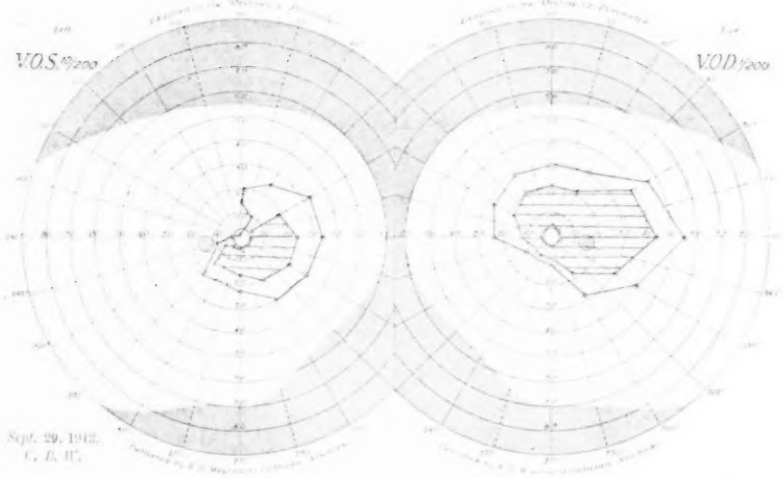

FIG. 46.—Case 14. Fields of September 29, 1913; plotted for $\frac{1}{4}$ sq. cm. discs only, showing further rapid progress, but with retention of macular fibres permitting of central fixation.

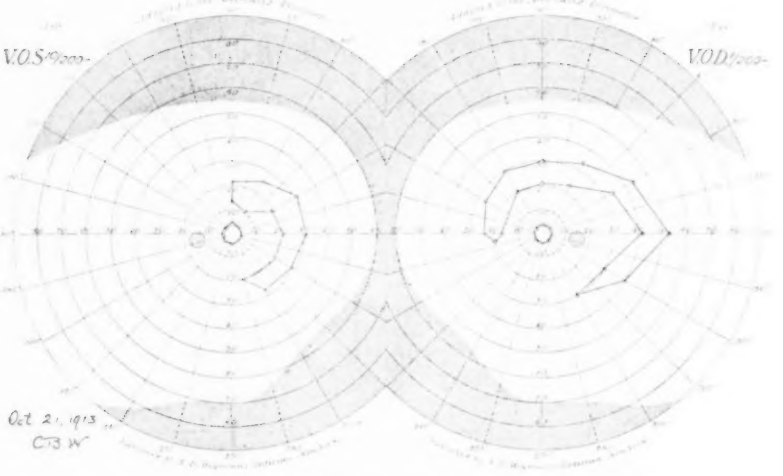

FIG. 47.—Case 14. Last fields of October 21, 1913, to $\frac{1}{4}$ sq. cm. discs, showing conditions before fatal operation. Fixation permissible owing to retained central vision.

November 17.—*Operation.* Transfrontal procedure with disclosure of irregular calcareous tumour, wrapped around chiasm. Attempt to dislodge the tumour caused bleeding. Post-operative hyperthermia. No return of consciousness.

November 21.—*Second-stage operation.* Transfrontal removal, with no anæsthesia, of large interpeduncular growth, causing unavoidable damage to interpeduncular tissues and death after twenty-seven hours.

Comment.—In this case, without the X-ray disclosure of a tumour, we should in all likelihood have been as uncertain of the diagnosis as our various predecessors have been.

It is notable that almost invariably in these cases of pituitary tumour (e.g., in all but one of the thirteen here recorded), even though pressure symptoms are pronounced, a swelling of the nerve-head does not occur. This we have attributed to the fact that the sheath of Schwalbe is so blocked as to prevent the crowding down of cerebro-spinal fluid around the nerve—in other words, to an interference with the physical conditions under which a choked disc is produced.

However, in a number of the cases in the series—of which Case 12 and Case 13 are examples—a low grade of œdema, occasionally with new tissue formation, has become superimposed on a primary atrophy, when intracranial pressure has become extreme and the nerve-sheaths have not been occluded by the growth. Usually, as in this particular patient, the choked disc is secondary to an internal hydrocephalus.

The so-called primary optic atrophy.—The comment on the foregoing case naturally brings up the question of a primary versus a secondary atrophy; and just how far the secondary changes may have been responsible for the irregular field deformations may be a matter of opinion. We have seen other examples of bizarre fields, most of them similarly associated with a normal sella and a congenital tumour, in which normal peripheries have been restored after a successful operation—in one case, indeed, after the constriction had advanced as far as in the last fields of this Case 13. In none of the cases in which prompt post-operative improvement occurred did the so-called primary atrophy show evidence of a superimposed œdema with its secondary new tissue formation.

We are aware that exception may be taken to our designation of the conditions of the nerve-head which we have observed, as "primary optic atrophy," for the reason that the subsequent return of vision in many of the disproves the presence of atrophy, on a strict

construction of the term. However, many of the patients have come to us after ophthalmologists elsewhere have given a similar pronunciamento. Doubtless if left to itself the condition in every case would tend in the long run, despite the occasional spontaneous recessions of which the process is capable and to which many have called attention [10], toward a terminal and permanent blindness. In a few of our own cases the history records periods of lowered visual acuity and contracted field peripheries from which there has been a temporary recovery, though after an interval vision has again failed. This of course means that the growth is capable of periods of increased tension for one cause or another (œdema, vascularity, &c.), just as a thyroid struma may enlarge and subside, though the tendency lies in the direction of a final increase in size despite the fluctuations.

Undoubtedly there may be other conditions which may cause a similar glistening pallor of the nerve-head, such as the toxic amblyopias from tobacco, lead, illuminating gas and so on, but we feel, in view of the far greater frequency of this appearance from a mechanical pressure against the nerves and chiasm, that doubt should always be put upon the toxic explanation of the process until a hypophysial source of the atrophy has been excluded. The diagnosis of lead, tobacco, and of syphilitic amblyopia had previously been made in several of the cases in our series, subsequently proved to be hypophysial.

It is often impossible to tell from the appearance of the disc whether or not there has been an irrecoverable destruction of the nerve. As mentioned in the comment on Case 9, dependence must usually be placed on the preservation of pupillary reactions and on the retention of some light perception, no matter how dim. We have seen vision return in blind eyes, though only once or twice in eyes with completely lost pupillary reactions.

This all goes to show what one of us has emphasized before, that demonstrable perimetric defects are more an evidence of a physiological block than of an anatomical destruction of the nerves in view of the great degree of recoverability in the transmission of light impulses on the release of the nerves from pressure. This is further supported by the fact that in several cases the histological examination of nerves has shown but a scattering of degenerated fibres, even though the ophthalmoscope had previously demonstrated the characteristic pallor attributed to advanced atrophy and the perimeter an advanced field defect. No correspondence has been found to exist between sharply cut quadrantal or hemianopic field defects and patches of degeneration seen on cross section of the optic nerves, such degeneration as might be

expected in accordance with Henschen's diagrams; for we have found that clean-cut functional defects in the fields may occur without any equally clean-cut fascicular disposition of degenerated fibres in the nerves.[1]

Recapitulation.

In a series of 454 cases classified as tumour of the brain there have been 101 in which the lesion was of hypophysial or parahypophysial origin, and in 81 of these cases chiasmal involvement led to deformations of the fields of vision. These deformations tended at the time of admission to be bitemporal in 26 cases, homonymous in 12, were unclassifiable in 8 cases, and the remaining 35 showed blindness in one or both eyes, making it difficult to tell in which group—bitemporal or homonymous—they belonged.

In cases of bitemporal hemianopsia it is unusual for the field defects to be bilaterally symmetrical, the text-book representation of the condition, with a vertical meridian dividing blind from seeing retina in each eye, being relatively uncommon.

In the majority of cases, whether of intra- or suprasellar tumour, the first perimetric indication of the process is shown by a slant in the boundary of an upper temporal form field and a corresponding quadrantal defect in the colour peripheries. For clinical convenience this is designated as Stage I of the process, and with a progressive lesion the field defects advance in characteristic fashion through Stage IV of actual hemianopsia to Stage VIII when blindness has ensued. In all stages the loss of colour perception is usually in advance of that for form, and the condition in one eye is usually in advance of that in the other, so that every possible stage combination in the two eyes may be met with.

When relief from pressure has been afforded by operative measures the recession of the defects takes place in a sequence, the reverse of that characterizing the stages of an advancing process. In the receding process relative paracentral scotomata often persist, as the functional vulnerability of the macular and paramacular fibres appear to be greater than that of the other fasciculi. Restoration of normal field peripheries is possible even when the process has advanced to or beyond the Stage IV of typical half vision, and some vision may be regained even after blindness has occurred, provided it has not been of too long duration.

In view of the prompt restorations in field outlines which may follow operative relief from pressure, it is evident that the so-called primary optic atrophy often does not represent an actual anatomical

[1] This matter will be made the special subject for further communication.

degeneration so much as a physiological block to the transmission of visual impulses.

In conclusion.—Detailed perimetry with small test objects of serial sizes, particular attention being paid to the shading off of the upper temporal peripheries and to the presence of relative paracentral scotomata in the same quadrant, is advocated for patients with pituitary disease in order that stages of hemianopsia antecedent to those usually recognized may be detected.

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THE DEMONSTRATION OF *SPIROCHÆTA PALLIDA* IN CHRONIC PARENCHYMATOUS ENCEPHALITIS (DEMENTIA PARALYTICA).

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In a previous issue of BRAIN [1] we discussed the relation of dementia paralytica to syphilis, and described how Noguchi and others had succeeded in demonstrating *Spirochæta pallida* in the brains of such cases. We mentioned that we ourselves had confirmed these findings, and since, so far as we are aware, there has been no detailed publication on the subject in this country, we now give illustrations of the cases referred to by us.

THE TECHNIQUE OF EXAMINATION.

The brains or fragments of brain were examined fresh, as received by post from Dr. Gettings, of the Wakefield Asylum, by the method of dark-ground illumination. The most satisfactory illuminator is the parabolic condenser of Zeiss, associated with a $\frac{1}{6}$ in. objective (correction collar), and a No. 6 compensating eyepiece. The best source of light is a small arc lamp the rays of which are passed through a spherical condenser filled with water. A small fragment of cortical grey matter about the size of a pin's head is removed from the specimen and broken up in a drop of saline in a watch-glass. The tissue should not be thoroughly emulsified since the specimen will then be too opaque for satisfactory examination. A small quantity of the fluid is placed on a slide and examined.

After five or ten minutes if the examination is negative a new area of the cortex should be taken, and thus a large area of the brain may be investigated. Wherever spirochætes were found that portion

of the brain was fixed in formalin and stained by Noguchi's modification of Levaditi's method for demonstrating spirochætes in sections. It is clear that the dark-ground technique gives no information as to the relation of the organisms to the surrounding tissues, but it very much facilitates the examination of sections, and almost ensures that spirochætes are present in the fragment selected.

The detection of *Spirochæta pallida* in sections of the brain is sometimes easy, but often extremely difficult. As will be seen from the accompanying exact drawings the organisms are at times degenerate or atypical, and if such an example is met with in a specimen in which the nerve fibrils have also taken up the silver, it is quite impossible to be certain that the object is a spirochæte. It is only when areas are found in which the fibrils are unstained, or when a focus contains typical as well as atypical spirochætes, that the diagnosis may be made with confidence.

We have therefore found that it is preferable to traverse a section all over rapidly, rather than to try to disclose single spirochætes in unfavourable areas. If this method is adopted, it will usually happen that a focus may be found in one portion of the slide only, and the spirochætes may be so numerous in this focus that they could not be overlooked. It is unusual to find a single spirochæte which is not accompanied by several others in the vicinity.

Notes of the Cases.

The following details of the cases include short abstracts of the clinical notes communicated by Dr. Gettings:—

Case 1.—W. G., male, aged 30. Duration of symptoms unknown; admitted June 13, 1912, died January 4, 1914. Clinical manifestations of dementia paralytica; grandiose delusions, speech and hands tremulous, pupils dilated and unequal.

Autopsy.—Great excess of subdural fluid. Brain weighed 1,590 grm. Pia increased in vascularity, thickness and opacity; large arteries atheromatous, grey matter dark and congested, granulations in fourth ventricle.

Microscopic appearances.—Slight cellular infiltration of the pia-arachnoid; general perivascular infiltration of the vessels in the cortex and in the medulla. Plasma-cells are relatively few in the infiltrations, the majority being large mononuclear cells (endothelial). In some places there is a slight occlusion of the small vessels.

Spirochætes.

Dark ground	...	Spirochætes very rare, found only in the frontal region.
Sections	...	None found.

Case 2.—M. A. H., female, aged 46. Duration of mental symptoms indefinite, said to have had a stroke in June, 1911; admitted August 20, 1913, died January 9, 1914. Pupils equal and contracted: patient had no knowledge of her surroundings or of what was said to her.

Autopsy.—Brain weighed 1,180 grm. Great increase of subdural fluid. Pia thickened, opaque, and adherent. Grey matter slightly atrophic but reddish in appearance, marked granularity of floor of fourth ventricle.

Microscopic appearances.—Marked infiltration of the pia-arachnoid with plasma-cells and lymphocytes; vessels appear to be normal. In the brain there is only a moderate perivascular infiltration, but in a few places it is very marked. No evidence of glial proliferation, pyramidal cells slightly degenerate.

FIG. 1.—Section from frontal region of Case 2 (M. A. H.). Zeiss $\frac{1}{12}$ and No. 6 compensating eyepiece. Noguchi's stain.

Spirochætes.

Dark ground ... Numerous *Spirochætæ pallidæ* in the frontal and central regions.

Sections... ... Large foci in frontal area, small in the central; spirochætes mainly superficial.

(*Vide* fig. 1.)

Case 3.—R. M., male, aged 35. Duration of mental symptoms indefinite; stopped work (coal miner) three days before admission; admitted August 3

1912, died January 13, 1914. Patient was quite demented, and had no knowledge of his surroundings.

Autopsy.—Brain weighed 1,140 grm. Considerable excess of subdural fluid. Pia-arachnoid thickened and opaque, granulations in the fourth ventricle, grey matter very atrophic.

Microscopic appearances.—Meninges here and there show a very slight cellular infiltration. In the brain no distinct perivascular accumulation of cells, though in places there appears to be a slight increase of these cells. The pyramidal cells show much atrophy and degeneration; slight increase of glial cells.

Spirochætes.

Dark ground ... One or two degenerate objects seen, but it was doubtful whether they were spirochætes or not.

Sections ... ... None found.

FIG. 2.—Section from frontal region of Case 4 (S. C.).

Case 4.—S. C., female, aged 40. Duration of symptoms, two months; admitted September 24, 1913, died January 13, 1914. Patient had no interest in her surroundings and was quite demented. She gave birth to a child on October 20, 1913, which, however, only lived a day. It had a diffuse cirrhosis of the liver with small-celled infiltration.

Autopsy.—Brain weighed 1,180 grm. Pia-arachnoid thickened and opaque, large arteries atheromatous, grey matter thin and congested, granulations in the fourth ventricle and lateral ventricles.

Microscopic appearances.—Meninges thickened and œdematous, slight infiltration with lymphocytes and plasma-cells, extravasation of red cells, proliferation of the endothelium of the vessels. In the cerebral substance the perivascular infiltration is very slight, the pyramidal cells show some degeneration but are normal in number.

Spirochætes.

Dark ground ... Spirochætes very numerous (two or three in each field) in the frontal and central regions.

Sections ... ... In places spirochætes very numerous.

(*Vide* fig. 2.)

Case 5.—J. T., male, aged 47. No previous history obtainable, admitted April 23, 1913, died January 17, 1914. Grandiose delusions: just before death had several convulsive seizures.

Autopsy.—Brain weighed 1,470 grm. Pia greatly thickened, opaque, and congested. Vessels showed patches of atheroma. On section grey matter yellowish and atrophic. Coarse granulations in fourth ventricle.

Microscopic appearances.—Meninges showed a moderate infiltration mostly of mononuclear cells, with a few plasma-cells. In the brain, general perivascular infiltration which included large numbers of plasma-cells.

Spirochætes.

Dark ground ... Spirochætes few in number and rather degenerate.

Sections ... None found.

Case 6.—J. T. B., male, aged 34. Duration of mental symptoms three weeks, admitted December 29, 1913, died January 6, 1914. History of syphilis. The clinical diagnosis was "cerebral syphilis," ptosis of left eyelid, left pupil dilated. Patient confused, violent, and resisted any attempt to examine him.

Autopsy.—Brain weighed 1,405 grm. Dura thickened and subdural fluid increased. Pia very vascular and adherent, grey matter congested, granulations along floor of fourth ventricle.

Microscopic appearances.—Marked cellular infiltration of the pia-arachnoid. The blood-vessels were engorged but their walls normal. Perivascular infiltration in the cortex was marked in places; plasma-cells were few in number. No obvious proliferation of neuroglia. Pyramidal cells were considerably shrunken.

Spirochætes.

Dark ground ... Spirochætes found in the frontal and central areas.

Sections ... None found.

Case 7.—A. R., female, aged 46. Duration of mental symptoms three months: admitted October 6, 1913, died January 8, 1914. Happy and fatuous; clinical diagnosis "dementia paralytica." Wassermann reaction positive in blood and cerebrospinal fluid.

Autopsy.—Brain weighed 1,140 grm. Enormous increase in subdural fluid, membranes slightly thickened, grey matter pale and atrophic, granulations in the fourth ventricle very marked.

Microscopic appearances.—Slight cellular infiltration of the pia-arachnoid. Vessels in the cortex showed only a moderate infiltration of their sheaths, though in one or two places it was more pronounced. Infiltration consisted of large mononuclears with a few plasma-cells. Pyramidal cells were decreased while glial cells were proliferated.

Spirochætes.

Dark ground ... Numerous spirochætes in the frontal and central and visual areas; most in the frontal regions.

Sections ... Several foci with numerous spirochætes in frontal area. (*Vide* figs. 3 and 4.)

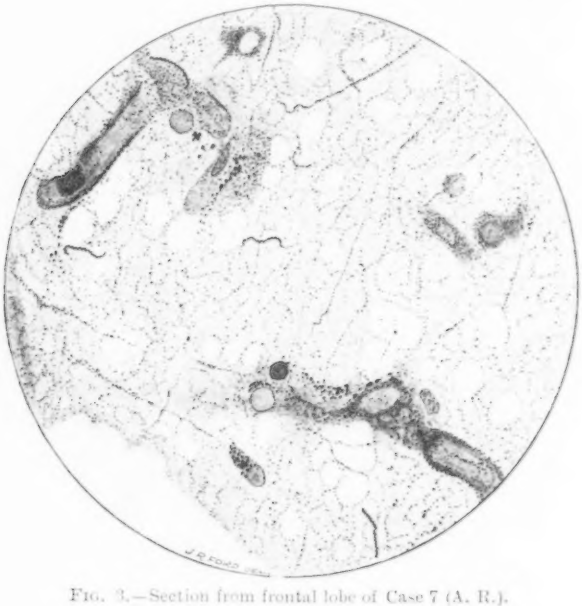

FIG. 3.—Section from frontal lobe of Case 7 (A. R.).

SUMMARY.

It will be seen that seven cases of "dementia paralytica" were examined, and that in six of these spirochætes were found by the dark-ground method, although when sections of these positive cases were cut the organisms were only detected in three. It is thus obvious that the latter method presents much greater difficulties than the former, the spirochætes being found only when they are numerous. Five cases also were examined in which the symptoms suggested a diagnosis of "dementia paralytica." This diagnosis was, however, found on section to be incorrect. No spirochætes were found.

These results do not illustrate the distribution of the spirochætes

over the whole cortex, because specimens were sent from a few regions only. It was, however, usual to find the organisms in all specimens sent when success was obtained with one.

All the cases examined had exhibited relatively acute manifestations before death, and the brains with one exception were not markedly wasted. The lesions on the whole appeared to be recent.

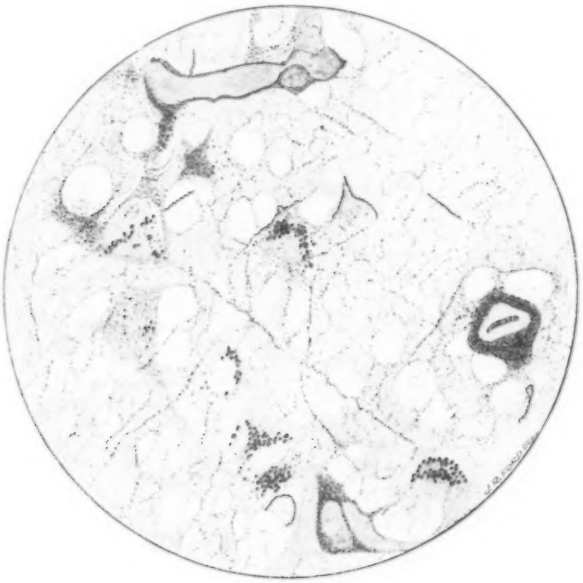

FIG. 4.—Section from frontal lobe of Case 7 (A. R.).

As already mentioned[1] the spirochætes were always confined to the grey matter; in one case only was a single specimen seen in the meninges. They were more or less superficial, and no constant relation could be observed between them and nerve-cells or blood-vessels. Some spirochætes indeed appeared to be applied to nerve-cells, but this distribution was not striking. Further no connexion was apparent between the organisms and cellular infiltrations; indeed they appeared to be scattered quite fortuitously about the superficial layers of the cortex.

It may be remarked that the drawings by Mr. J. R. Ford give an extremely accurate impression of the appearance of the sections under the microscope.

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SOME CASES OF A "CROSSED REFLEX" ASSOCIATED WITH PAIN: THE BEARING OF THE CROSSED REFLEX UPON THE THEORY OF THE EXISTENCE OF AUTOMATIC SPINAL CENTRES.

BY E. D. MACNAMARA AND E. B. GUNSON.

THE five cases, the notes of which are subjoined, are, in the main, presented for record for two reasons; firstly, that they afford examples of a reflex of rare occurrence, and secondly, because the phenomenon of pain which occurred on the application of the stimulus eliciting the reflex in four of the cases has not, so far as we are aware, been previously described. The cases are also presented as data for the further elucidation of the problem as to whether such movements as are described may be regarded as evidence for the existence of automatic spinal centres.

Case 1.—The patient, a male, aged 15, was admitted to the West End Hospital for Nervous Diseases on May 16, 1912. Two months before admission he had suffered from loss of speech, loss of memory and mental dulness. These symptoms had lasted for fourteen days and the patient then completely recovered. He remained well for four weeks, when the symptoms recurred. On admission the history was that the patient had not spoken for a week, and that he had vomited once before breakfast. He took very little notice of his surroundings, and his face was expressionless. He appeared to understand what was said to him, and carried out simple instructions. He gave his name and address correctly, but showed no further inclination to speak. On examination there was no evidence of loss of power or of sensation. There were no abnormal movements, and co-ordination was good. There was well marked optic neuritis in the right eye. The superficial reflexes were very active, and equal on the two sides. The supinator, knee- and ankle-jerks were brisk, and equal on the two sides. There was a tendency to ankle-clonus on the right side. Oppenheim's and Gordon's signs were present on the left side only. Incontinence of urine had occurred on two occasions.

The boy gradually became more dull, and entirely refused to speak. The optic neuritis became more marked on the right side, and also gradually began on the left. It was thought possible that there was a cerebral tumour affecting the right frontal region. On June 6 Mr. Ballance performed

craniectomy over this region. There was no obvious increase of intracranial pressure. The dura was opened, but no tumour was found. After the operation the patient became very suspicious, resented all efforts made on his behalf, and frequently refused food.

It was now first noticed that on grasping firmly the quadriceps extensor muscle with the thumb and fingers immediate flexion at the opposite hip-joint took place. The reflex was equally present on each side. No response was produced by merely stroking the thigh or by percussion of the muscles or bones. There was no movement at the hip-joint of the side grasped, though dorsiflexion of the great toe on this side did occur. It was a particularly remarkable fact that elicitation of the reflex apparently caused considerable pain, and was much resented by the patient.

The patient was discharged from hospital about one month later. By that time his symptoms had all cleared up, and the crossed reflex had disappeared. The swelling of the discs had very largely subsided. He remained well until December, 1913, and presented no symptoms or abnormal physical signs, save that the discs suggested a past papillo-œdema. At the beginning of December, 1913, he began to be silly and to do stupid things. He was forthwith admitted to the hospital, and soon showed the symptoms of negativism which had been so marked before. The cross reflex reappeared, but the movement was of smaller excursion and was unattended by pain. There were no other abnormal physical signs, and the papillæ were not more swollen. The symptoms gradually abated, and the patient was quite well by the middle of January, 1914. At no time during his illness had anything abnormal been discoverable in any of his other organs.

Case 2.—The patient, a male, aged 29, was admitted to the West End Hospital for Nervous Diseases on September 2, 1912. This patient had, according to his history, never been intellectually very bright. He had contracted syphilis four years previously. In May, 1908, he had an attack of generalized convulsions. He was semiconscious for about two weeks, and during that time had a series of epileptiform attacks. He was then put on bromide, and remained free from fits for some ten months. The fits then recurred, and he has had them occasionally ever since. In 1910 an operation was advised, but was refused. X-rays at that time showed thickening in both frontal regions, but no tumour mass. In February, 1912, a positive Wassermann was obtained, and patient was dosed with salvarsan, and again in May and June. He also had inunctions of mercury. During the last four years he had had frequent and severe frontal and occipital headaches and occasionally had vomited, without other dyspeptic symptoms.

On admission to the hospital the patient was dull and lethargic, with occasional choleric outbursts. His memory was poor, and he was conscious that it was failing. He could not detect strong smells through either nostril. A double papillo-œdema was present, and the discs also suggested an early stage of atrophy. He could perceive acids on both sides of his tongue, but could not taste sugar or salt. So far as the rest of the body was concerned there appeared, on the sensory side, to be no loss of any faculty for the

perception of any sort of stimulus. Stereognosis was also good. On the motor side there were no marked abnormal physical signs. There was no weakness, wasting, spasticity or flaccidity, but there was some inco-ordination affecting the upper extremities. The knee- and ankle-jerks were exaggerated equally on both sides. There was no ankle-clonus. The abdominal reflexes were absent on both sides. The plantar reflexes were flexor. On grasping the quadriceps extensor mass on one side slight but definite flexion occurred at the opposite hip, and was most evident on the right side. A remarkable feature was that the patient complained of the pain caused by grasping the quadriceps. There was occasionally incontinence of urine. At this time the Wassermann reaction was negative. In the cerebrospinal fluid 1·3 lymphocytes were counted per cubic millimetre. A fit occurred at this time, and it was noticed that the left arm first became rigid in extension, and that the head and eyes turned to the left before the convulsion became generalized. On November 26 the patient died, and on *post-mortem* examination a subcortical cystic glio-sarcoma was found in the upper part of the right frontal lobe extending back almost to the Rolandic area.

Case 3.—The patient, a male, aged 15, was admitted to the General Infirmary, Leeds, on May 10, 1913. In May, 1912, the patient began to suffer from severe frontal headache, with vomiting, shortly after food. General malaise and constipation were associated with these symptoms. They persisted for a week, and then completely disappeared. A similar attack occurred seven weeks later, and lasted two weeks. A third attack occurred four weeks after the second, and followed the same course for a week, when the patient was seized with a severe cramp affecting the right foot, spreading slowly upwards until the whole of the right side was involved. The cramp was accompanied by weakness, and after lasting for an hour passed off in inverse order. There was no loss of consciousness. The following day the other symptoms subsided. A fourth and fifth attack occurred, at intervals of a month, like the third, and associated with cramp and weakness of the right side, each attack lasting about one week. A sixth attack occurred in February, 1913. With the onset of the cramp the patient became dazed, and was led to bed. He became semi-conscious, his eyes became fixed, and he screamed out frequently. On the following day the symptoms had subsided. The last attack, five weeks before admission, was very slight, and was accompanied with cramp and some transient weakness of the left leg. In the intervals between the attacks the patient had followed his employment and enjoyed good health.

On admission the patient presented no symptom of mental disturbance. Double optic neuritis was present, and was more marked on the right side. On the sensory and motor sides he presented no abnormalities. The supinator, knee- and ankle-jerks were present, and were equal. The superficial reflexes were present and were active, and equal on the two sides. The flexor response was present on the two sides. On grasping the left quadriceps extensor mass slight flexion occurred at the right hip and slow extension of the great toe on the right side. There was no movement of the other toes or at the left hip.

On grasping the right quadriceps there was no response on either side. The provocation of the reflex on the left side caused considerable local pain; stimulation of the right side was quite free from pain. There was no symptom connected with the sphincters. Examination of the other systems revealed nothing abnormal. The patient remained in the hospital for seven weeks, during which time the optic neuritis completely subsided, and there was no return of symptoms.

Case 4.—The patient, a female, aged 10, was admitted to the General Infirmary, Leeds, on June 12, 1913. The patient had not been well for six months. Two weeks before admission there had been headache, giddiness and occasional vomiting. Ten days before admission an attack of convulsions occurred affecting the face and the upper and lower extremities. Since that time the patient had remained dazed, and had not spoken or taken notice of her surroundings.

On admission the patient was drowsy, but when roused understood what was said to her and performed simple movements. She did not speak. On examination an external strabismus was found on the right side and an internal on the left. Double optic neuritis was also present. There was paresis of the lower part of the right side of the face. There was very little voluntary movement of the limbs, which were rather flaccid. The abdominal muscles were also relaxed. There was, however, some degree of rigidity about the neck. The supinator jerks were present. The knee- and ankle-jerks were absent. Kernig's sign was present on both sides. The abdominal reflexes on the right side were active, but on the left were very feeble. Oppenheim's, Gordon's and Chaddock's signs were absent on both sides, except that Oppenheim's was occasionally present on the right side. On grasping the right quadriceps extensor mass there sometimes occurred slight flexion at the left hip, while there always occurred dorsiflexion of the right big toe and frequently also dorsiflexion of the left big toe. On grasping the left quadriceps mass no movement was produced at either hip, but there was always dorsiflexion of the right big toe, and sometimes dorsiflexion of the left big toe. Elicitation of the movement on either side caused great pain. The patient died on June 30, and on *post-mortem* examination extensive tubercular meningitis was found. The cerebellum was also found to be the seat of a few minute scattered tubercles.

Case 5.—The patient, a female, aged 8, was admitted to the West End Hospital for Nervous Diseases on March 6, 1913, for paralysis of both legs dating from birth. The patient had been a full-time child, and her birth had been normal. She had never been very intelligent, and has never talked properly. She had never walked or even stood up, and assisted progression was of the cross-legged type. The sphincters have always been well under control.

On examination there was found to be some degree of spasticity of both legs and of the right arm. Microkinetic movements of all the limbs were present. The tendon-jerks were all exaggerated, and both plantar reflexes

were of the extensor type. A Foerster's operation was performed by Mr. Laming Evans on June 24, and the spasticity of the limbs became slightly less. In this case tickling the sole of the left foot produced a movement of flexion of the right thigh, while tickling the sole of the right foot produced no movement. Grasping the muscles of the calf or the front of the thigh of either leg produced flexion of the right thigh but not of the left. In this case there was no associated pain.

To emphasize certain salient points in the histories of these cases we have tabulated them.

No. of case	Place at which stimulus was applied and character of stimulus	Resulting movement	Whether pain was present or not	Whether extensor response as elicited by ordinary methods was present	How case terminated	Character of lesions
1	Grasping the quadriceps extensor mass on either side	Flexion at opposite hip and dorsiflexion of great toe on the side stimulated	Pain was present	No	Symptoms cleared up	? lesion of right frontal region.
	Stroking the skin	No movement				
2	Grasping the quadriceps mass on either side	Flexion at opposite hip. No movement of the big toes	Pain	No	Death	Tumour right frontal region.
3	Grasping quadriceps mass on left side only	Flexion of opposite hip and dorsiflexion of opposite big toe	Pain	No	Symptoms cleared up	?
4	Grasping quadriceps mass on right side	Flexion of opposite hip and dorsiflexion of right big toe and occasionally of left big toe	Pain	Extensor response elicited by ordinary methods	Death	Extensive tubercular meningitis.
	Grasping quadriceps mass on left side	No movement at hips, but dorsiflexion of right great toe and occasionally of left				
5	Grasping the quadriceps mass or the calf on either side	Flexion at right hip. There was no movement at the left hip	No pain	Extensor response elicited by ordinary methods	Symptoms remained much as they had always been	Double cerebral sclerosis.
	Tickling the sole of the left foot	Flexion at right hip				
	Tickling the sole of the right foot	No movement at either hip				

An analogous reflex has been described by Levi-Bianchini [3]. According to him, in certain cases of mania, if the muscles of the calf

be squeezed on either side of the tibia, as the patient lies in bed, there results an immediate and energetic contraction of the quadriceps femoris. He writes that in most cases the reflex may be obtained on both sides; in others, however, it can only be obtained on one side. Gordon [1] had previously described an extension of the big toe occurring in patients with damaged pyramidal tracts on pressure being applied to the muscular mass constituting the calf. Guillain [2] has reported the presence of a contralateral flexion of the thigh upon the abdomen, and of the leg upon the thigh with abduction of the limb upon compression of the quadriceps femoris in meningitis of varying etiology. Marie and Foix [4] described movements, in certain cases, additional to those observed by Gordon, in which, by pressure upon the calf the great toe extended, the foot dorsiflexed upon the leg, the leg flexed upon the thigh, and the thigh upon the abdomen. Occasionally, also extension of the other leg was noticed. In cases of compression paraplegia, of disseminated sclerosis and of syringomyelia, the same observers have observed that on forcible flexion of the toes the same movements of shortening of the leg are produced, and may be followed by extension of the other leg. This movement appears to be analogous to the "stepping reflex" described by Sherrington [10] and Philipson [8] as occurring in animals, and which has otherwise been called the "crossed extension reflex." Marie and Foix conclude that such complex movements in fact represent the automatic movements of walking, that the elementary movements of walking can, by excitation here and there of the leg, be resolved into isolated muscular contractions, and that this automatism exists normally but becomes exaggerated by certain pyramidal lesions. They are further of opinion that Babinski's sign belongs to the totality of the complex automatic movements which together constitute the mechanism of progression, since at the moment when in walking the leg is, in part, flexed in the act of raising it from the ground the great toe is dorsiflexed. The sign is described as the minimal reaction to excitation.

These views are not universally accepted. V. Woerkom [11] lays stress on the sort of stimulus applied to arouse the reflex movement. For instance, the passive flexion of one limb, in which there is no association of pain, provokes the extension of the other (as, he says, is proved by the experiments of Freusberg and Sherrington), whereas a painful stimulus does not produce contralateral movements (and in fact it inhibits them), but rather a simple movement of withdrawal from the stimulating agent. He points out, however, that the contracting

muscles are the same for the movement of withdrawal as for that phase of walking which consists in the removal of the foot from the ground. To explain the mechanism of the extensor response v. Woerkom argues from certain ontological predicates. The big toe early in its development is adapted to perform prehensile functions. The position for prehension is scarcely compatible with comfortable or efficient progression. To overcome this incompatibility the extensor longus hallucis comes into action, for it has abductor as well as extensor functions. This function is not shared with the extensors of the other toes, and later on in life, when the art of walking has been acquired, is lost. In the process of adaptation to walking, which is presumed to have occurred when our ancestors exchanged arboreal life for one passed on the ground, the muscle has acquired an extensor function which is no longer a useful one when progression and not prehension becomes the important work of the toes. For progression the big toe acts with its fellows, for prehension in opposition to them. In babyhood, before progression is necessary, the big toe acts, on stimulation of the plantar surface of the foot, in opposition to the other toes; and similarly in organic maladies of the nervous system this ancient function again appears. To quote v. Woerkom's own words the movement is, "The vestige or revival of a lost function which consisted in the adaptation of the individual's foot to progression on the flat, and in which the great toe was still capable of taking a position of opposition to the other toes." Marinesco and Noica [5] reach the conclusion that normal subjects do not react, in respect of painful excitations, as do those suffering from spastic paraplegia who have almost completely lost the power of voluntary movement, whether they have or have not preserved sensibility to pain. In the normal individual movement is always in the direction of removing the limb from the disagreeable or painful excitation. This is not so of the movements which occur in spastically paraplegic individuals, which have not a character suggestive of flight from danger. The movements need not be accompanied by sensation, and can therefore be provoked unknown to the patient. Further the range of the movements diminishes if the limb be rendered anæmic by an Esmarch's bandage, and is exaggerated when blood is readmitted into the limb. On the other hand, true movements of defence are exaggerated when the limb is rendered anæmic, and are associated with much pain. Rossenda [9] relates a case of tabes in which all the tendon and bone reflexes were absent, as were also the cutaneous reflexes, including the plantar as ordinarily provoked or evoked. If, however, the

sole of the foot were forcibly stroked with the handle of a percussor, or if the toes were overflexed, certain movements occurred. The stroking of the sole and the flexion of the toes were not felt as such, though associated both with the stroking and the flexion there was much pain, which the patient compared to the lightning pains he occasionally experienced at other times. The movements consisted in energetic flexion of the thigh upon the trunk, of the leg upon the thigh, and of the foot upon the leg. While these movements of flexion went on extension of the opposite limb occurred, provided it had beforehand been placed in an attitude of flexion upon the abdomen. Rossenda is of opinion that, conformably to the views of Marie and Foix, the retraction which in certain pathological conditions occurs in consequence of excitation of the sole of the foot or of the joints of the toes, and which may or may not be associated with extension of the other leg, should be considered as due to cord automatism and not as a simple reflex. In a further communication Marinesco and Noica [6] arrive at the conclusion that the phenomena which occur on the side excited, or by propagation on the opposite side, vary with the intensity of the excitation and secondarily with the position of the limbs before excitation. In other words, these authors conclude that there does not exist in the cord a co-ordinating motor centre having some degree of independence in the choice of movements, or that if such a centre exists it is absolutely subordinate to the intensity of the excitation, and to the position of the limbs at the moment of excitation. Pastine [7] does not think that the extensor response can be placed among the automatic movements of walking, and that it occurs equally with movements, voluntary or reflex, of extension of the lower limb.

It will be noticed that in every one of our cases grasping the muscular mass on the front of one thigh resulted in a movement of flexion at the opposite hip-joint; that is a "crossed reflex" was present. This movement of flexion at the hip was, in Case 1, associated with dorsiflexion of the great toe of the opposite side to that upon which flexion at the hip occurred, that is the dorsiflexion of the toe took place on the side to which the stimulus was applied. In Case 3 the dorsiflexion of the great toe and the flexion at the hip-joint took place on the same side, that is on the side opposite to the one stimulated. In Case 4 dorsiflexion of the great toe on the side opposite to the one stimulated always took place, and dorsiflexion of the great toe on the side stimulated occasionally took place, a condition of affairs combining the characteristics of Cases 1 and 3. In Cases 2 and 5

no movement of the toes was noted. It appears to us difficult to regard a crossed reflex as a merely defensive one, presuming always that neither limb is by any sort of palsy deprived of the power of movement. It is, of course, the case that a movement of withdrawal of a limb occurs when any part of it is unpleasantly stimulated, unless such movement is inhibited from higher centres. It may be argued that such movements of withdrawal persist as habits and may at length involuntarily occur as reflex movements when the limb is stimulated not unpleasantly. It cannot, however, be so readily assumed that a movement of the limb opposite to the one stimulated associated with no movement of the stimulated limb is a movement of mere withdrawal or of defence. Indeed, supposing the motility of the stimulated limb to be unaffected, it is hard to offer any explanation other than that such a movement is one of the series of movements involved in progression and may, therefore, be taken for what it is worth, as supporting the hypothesis of the existence of automatic spinal centres. Considering in addition to the crossed movement of flexion at the hip the associated movement of the big toe, occurring in Cases 1, 3 and 4 of our series, we find ourselves in a difficulty in dealing with this problem, since in Case 1 the movement of the big toe was not on the side of the flexion of the hip, in Case 3 it was on the side of the flexion of the hip, and in Case 4 it was sometimes on the side of flexion and sometimes on the opposite side. From such a jumble of occurrences in so small a number of cases it is hard to draw any inferences, but we doubt if any that might be drawn could be made to support the hypothesis of the existence of spinal centres. It is true that the movement of toe and thigh in Case 3 might be harmonized and regarded as simultaneous movements of progression, since while the thigh is flexed upon the trunk as it is being brought forward in progression the great toe tends towards extension, in order, as we think may fairly be presumed, to better clear the ground. But such an explanation will not fit Case 1, for while a thigh is moving forward in progression the great toe of the opposite foot tends towards a movement of flexion, applying itself to the ground preparatory to the shove off of the foot from the ground. If the movement in Case 3 was normal to an automatic act of walking the movement in Case 1 was not. In Case 4 movements of the toes occurred indifferently, and cannot be cited in favour of automatism. It will be noted, however, that in Cases 1, 2 and 3 the extensor response could not be elicited by the ordinary methods employed; it did not occur as part of a movement of withdrawal from stimulation; and this fact, coupled with the occurrence of the movement simultaneously, with flexion of a thigh,

suggests to us that the movement might be regarded rather as part of automatic progression than as one of defence. In short, facts from these cases may be adduced in support of either hypothesis. Marie and Foix (*vide supra*) regard an extensor response as a minimal reaction, the complete reaction containing in addition to the movement of the toe flexion of the thigh. We would point out that, if this be so, we obtained in Cases 2 and 5 a reaction in which the minimum did not occur, but in which another feature of the reaction did occur. We think that the inference must be that it is incorrect to speak of the extensor response as a "mimimal reaction," since we should in that case have a state of affairs in which the greater did not contain the less. We believe, accepting for the moment the existence of the automatic spinal centres, that we are only justified in regarding one movement as co-ordinate with the other; the extensor response occurring commonly and the complete crossed reflex but rarely.

We desire particularly to draw attention to the very remarkable association of pain with the occurrence of the reflex in Cases 1, 2, 3 and 4. The amount of pain appeared wholly disproportionate to the character of the stimulus applied. In a normal individual such a stimulus produces no pain whatsoever and in several hundred patients suffering from a variety of maladies we have failed in applying a like stimulus to elicit pain. The pain was located at the site of the place where the muscular mass on the front of the thigh was grasped. In Case 1 the site of the lesion was supposed to be the right frontal region. In Case 2 the site of the lesion was the right frontal region. In Case 3 no particular locality could be assigned to the lesion. In Case 4 the lesion was a diffuse one and in Case 5 there was a presumption in favour of extensive cerebral sclerosis.

We are indebted to Drs. Wardrop Griffith and Maxwell Telling for permission to record Cases 3 and 4 respectively.

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A CASE OF MOTOR DYSPRAXIA AND PARAPHASIA: AUTOPSY: TUMOUR IN SUPRAMARGINAL CONVOLUTION.

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ALTHOUGH the manifestations which are included under the term motor dyspraxia are found not infrequently in cases of hemiplegia, aphasia and other cerebral disorders, the number of cases which have been fully investigated both clinically and pathologically is still small. Moreover, in the majority of cases which have been reported in the literature motor dyspraxia has been one only of a series of manifestations, and at necropsy multiple lesions have usually been found. In the patient who forms the subject of this note the clinical interferences were limited to motor dyspraxia, dysgraphia, paraphasia, and amnesic aphasia, and at autopsy a small, well localized, secondary carcinomatous nodule in the left supramarginal convolution surrounded by recent cerebral softening was found. The case, although incomplete from a pathological point of view owing to the fact that a detailed microscopic investigation of the fibres destroyed by the tumour was impossible, seems to be one of considerable importance for the localization of these interferences in the cerebral hemispheres.

Wilson [6] defines *motor dyspraxia* as "the difficulty in performing certain subjectively purposive movements, or movement complexes, with conservation of motility, of sensation, and of co-ordination." Liepmann [3] terms the difficulty in finding words *amnesic aphasia*, and gives the name *paraphasia* to the difficulty with speech characterized by "omissions, duplications, perseveration to earlier syllables, anticipation of later syllables, sliding off into words related in sound and sense, fusion of parts of suitable words with those of similar sound and similar sense, and loss of sequence in words owing to the action of accidental sensory impressions."

Whilst under observation from a clinical point of view the patient was for twenty-one days a satisfactory subject, and during this time

his condition altered little. Twenty-four days before death, however, weakness of the left half of the body and hebetude appeared, probably owing to the development of cerebral softening around the tumour.

At the International Congress of Medicine in London in 1913, Liepmann [3] said, "the region which is by far the most frequently affected in well developed apraxia is the left parietal region. This I have proved by a study of twenty-six cases of my own and of the whole of the literature on apraxia. Indeed there are sufficient cases of apraxia in which only lesions of the parietal lobes have been found." Kleist [1] in 1911 analysed the findings at necropsy in twelve recorded cases of ideo-kinetic dyspraxia, such as this patient exhibited, and found that in all lesions of the supramarginal convolution had been present; in eight of these other lesions also existed. In a case reported by Strohmayer [5] in 1903 severe motor dyspraxia affecting all four extremities, paraphasia, loss of initiative, and considerable difficulty with reading and writing were associated, and at autopsy a cyst 5 cm. in diameter was found in the left supramarginal convolution. Isolated lesions of vascular origin were also found in this region in two cases reported by Kroll [2] in 1910 and in one reported by von Stauffenberg [4] in 1911. These four cases all showed clinically severe motor dyspraxia of all the extremities and both halves of the trunk, difficulty with reading, inability to copy, difficulty in writing to dictation, paraphasia, and considerable loss of memory; and at autopsy cyst formation or cerebral softening consequent on interferences with the blood supply of the supramarginal region on the left side was found in each. Von Stauffenberg [4] sums up his conclusions "lesions of the lower portions of the parietal lobe which do not involve either the angular gyrus or first temporal convolution, but affect the subcortex almost down to the lateral ventricle, regularly give rise to apraxia and an interference with the mechanism of speech, characterized by paraphasia and gross interferences with reading and writing but do not affect the understanding of speech." The findings at autopsy in my case confirm his statement that "disease localized to the supramarginal convolution on the left side can cause apraxia." Nowhere in the literature, however, have I been able to find any record of a case where a well localized neoplasm, either primary or secondary, has given rise to motor dyspraxia and paraphasia.

In this case it is also noteworthy that, although from the appearance of slight proptosis, paralysis of the external rectus and paresis of the internal rectus muscles, and the distorsion of the outlines of the sella turcica as seen in the radiogram, the presence of secondary carcinoma

in the region of the pituitary gland was diagnosed during life and its presence confirmed at *post-mortem* examination, no signs of superpituitarism or subpituitarism were present; yet, as seen in the microscopical specimens, practically the whole of the three lobes of the pituitary gland were infiltrated by growth and little normal glandular tissue could be discovered.

Sidney T., married, engineer's fitter, aged 41, was admitted to the London Hospital under the care of Dr. Lewis Smith, on August 17, 1914, and died on September 29.

Between 1903, when he married, and 1913 his health was uniformly good. About February, 1914, he began to complain to his wife that his habitual skill with his tools was failing; he became depressed and irritable, but his health remained fair until June, 1914. During the last week of that month his left wrist swelled, and the skin over it became red, shiny, and tender. He saw his local doctor, who diagnosed gout and kept him from work for seven days. Three days after his return to work he developed a left-sided headache. The headache was severe, persistent, and kept him awake at night time; in bed he was restless and tossed about. On July 10 he began to "see double"; he then attended at an Eye Hospital, where he was found to have paralysis of the left sixth cranial nerve and a certain amount of proptosis on that side. During the month of July, speech was not affected, memory was good, and yet he "thought he was going out of his senses," and became more irritable and more depressed. About 10.30 p.m. on August 3, he had a seizure; "for a minute or so he went stiff, his eyes rolled, he became black in the face and foamed at the mouth." After the seizure he complained that his headache had increased, he tossed about but only partially roused. Half an hour later, about 11 p.m., he had a second similar attack lasting a minute or so. He then remained unconscious until 12.45 a.m. on the 4th, when he suffered from a third similar, but more severe, attack in which he passed water. For the next eight hours he lay drowsy or asleep, and did not know where he was. On August 4 the pains in his head were extremely severe, diplopia had become more troublesome and speech had altered. He was kept in bed from the 4th until the 9th, and gradually, despite the pains in his head, "seemed to become his old self again."

During the week, August 10 to 17, he gradually "went downhill." His memory failed and "he would wander and lose his words, although his mind was otherwise clear." His wife said that he seemed to be unable to recognize that he was only addressing her as soon as he began to talk about other people's affairs; "when talking at home to me alone, he would speak of my wife Annie just as if he were in company." On the 10th, for instance, he was talking to his wife about the affairs of his brother-in-law, when he suddenly remarked "now my wife Annie cannot help me"; on the same day he was found gazing into a cupboard, and when his wife asked him what he was doing he remarked "I don't know why I look in there, I must be mad."

On the 16th he lay in bed all day complaining of intense pain in the left side of his head.

He denied all venereal infection, and the Wassermann reaction both in the serum and cerebrospinal fluid was negative. At the age of 17 he had suffered from diphtheria. His wife, who was alive and well, had had two children, born in 1904 and 1907, who survived and were healthy.

The patient was a well developed, pale, thinly covered, right-handed man of 41, showing signs of recent wasting. On admission the temperature tended to be slightly irregular and varied from 97° to 99° F. The apex beat of the heart was felt in the fifth space just inside the midclavicular line. The area of cardiac dulness was not increased, there was no cardiac displacement, and the heart sounds were clear. The wall of the radial artery was just palpable, but the tension was not raised. The rate of the pulse varied from 60 to 80 beats per minute. The liver and spleen could not be felt and to percussion were not enlarged. The quantity of urine passed in twenty-four hours varied from 28 oz. to 56 oz.; albumin and sugar were never present. The lymphatic glands above the clavicles and in the axillæ and groins were not enlarged. The fingers were slightly clubbed.

For some days before his admission he had complained of a dry, hacking cough, but had not expectorated. The chest was symmetrically developed, but moved badly in its upper half on the left side. The movements of the lower portions of the chest on the two sides were equal. Tactile vocal fremitus and vocal resonance were unaffected. In front, the percussion note was impaired in the second, third and fourth interspaces on the left side, but was almost normal in the supraclavicular fossa; behind, the note was flat over the upper part of the infraspinous fossa and between the second and fifth thoracic spines and the vertebral border of the scapula, and was impaired over the whole of the upper lobe on the left side. The breath sounds over this lobe were weaker than over the corresponding areas on the right side, and from time to time fine or medium crepitations were heard there. A radiological examination of the chest at this time showed an opacity of almost uniform density of the whole of the upper part of the left side, with displacement of the trachea to the right, but no displacement of the heart. A radiographic plate in addition gave evidence of some consolidation in the region of the root of the lungs. The rate of respiration varied from 20 to 26 per minute.

The knee-jerks were equal and readily obtained. The ankle-jerks were normal, there was no ankle-clonus, and both plantar reflexes gave a flexor response. The wrist- and elbow-jerks were unaffected, and the jaw-jerk was obtained. The abdominal reflexes could be elicited, but were sluggish. The ocular and pharyngeal reflexes were normal. The sphincters were under control, and the movements of the spine were unaffected.

To sensory testing no loss of sensibility was discovered. The light touches of cotton-wool were everywhere appreciated, and could be localized. The pricks of a pin and the tests with hot and cold tubes at varying temperatures elicited accurate replies. The vibrations of a large tuning-fork (C = 128) were

well recognized on all four extremities, and the duration of their recognition was nowhere shortened. Recognition of posture and passive movement was unaffected, and without much effort he could identify and give the properties of objects placed in his hands. There was no falling away of the outstretched upper extremities on closure of the eyes.

In the extremities and trunk there was no local muscular wasting, and no change in muscular tone. Movements of normal range and power were possible at all joints, and the patient did not complain of spontaneous twitchings, and showed no other involuntary movements. Gait was little affected, and Romberg's sign was not obtained. The grasps of the hands were powerful and equal.

He complained much of diplopia. Movements of the right eye were well carried out. The left eye was turned inwards. With this eye he experienced considerable difficulty in looking inwards to the right, and was completely unable to look outwards to the left. The left eyeball was more prominent than the right, but there was no ptosis of the upper eyelid, and nystagmus was not present. The pupils were equal, and reacted briskly to light and to accommodation. Facial movements were normal, and the movements of the palate and larynx were unaffected. The trapezii and sternomastoid muscles acted normally, and he suffered from no difficulty in swallowing. The tongue was protruded straight, and could be held steadily.

Smell, taste, and hearing were unaffected. Vision for form, colour, &c., with both eyes was normal, and the visual fields for white and colours were not diminished. On the left side the vessels of the ocular fundus were full and tortuous, and the lamina cribrosa could not be distinguished; on the right the vessels were full, the edges of the disc blurred, and the lamina cribrosa filled. The swelling of the disc, however, measured less than 1 D.

He constantly complained of pains in the frontal region, and said that they were always worse on the left side than on the right. When asked where the pains were situated, he put his left hand over the left orbital and temporal region, and swept it round over the malar bone towards the left ear. He said that the scalp here was tender, and pressure over the left orbit and temple elicited a complaint of "pain." The pains were usually worst in the early morning and after sleep, but were constantly present. He never complained of pain in the occipital region. He said that the pains in his head made him feel sick, but whilst under observation never vomited. On August 3, 1914, he had suffered from three epileptiform attacks; after this date until death he was free from seizures.

He was attentive and, except when the pains in his head were severe, took considerable interest in the happenings of the ward; he was, however, never interested in his fellow patients. He was extremely grateful for the trouble taken in the investigation of his case, and willingly undertook every test. When his headache was severe, he lay drowsy and inattentive, but when addressed would always rouse himself and attempt to answer questions.

Throughout his stay in hospital memory seemed to vary; on some

occasions he could recall events and give details of his illness, work, and past life; on others all efforts of memory seemed impossible. At times he would describe spontaneously the recent events which had happened around him, and on these occasions gave evidence that he could retain recent impressions.

In space and time he was well orientated; he knew the day, hour, and manner of his arrival at hospital, admission, and of the various examinations. In general he was cheerful, but was easily moved to laughter or tears; when the pains in his head were severe, he was irritable, suspicious, and depressed. Except in the matter of words he was not very subject to suggestion. The last phrase of a sentence seemed frequently to be dominant, and he would often repeat several times the last few words made in answer to a question, even though he was not further addressed.

Judgment and the power of reasoning seemed little impaired, despite his difficulty in expressing himself. He associated ideas readily and could appreciate humorous episodes and the point of a story. He was not subject to hallucinations, and never suffered from delusions. He did not complain of worrying dreams or nightmares. He slept well, and, for the greater portion of the twenty-four hours, when left to himself, lay drowsy or asleep.

In eating and dressing himself he was careless, but became annoyed when he dropped food or otherwise dirtied his bed.

He rarely moved from his position in bed, and, except when directly addressed, rarely spoke. Individual words were well pronounced. He could put together spontaneously short reasonable sentences; in each sentence, however, some words were always badly articulated and slurred. Phonation was monotonous. In attempting to give an account of himself such remarks as: "Last Easter, that was in nineteen, let me see, let me see, fourteen, last Easter, last Easter, to the best of my recollection, as far as I can remember, I went to Easter, to Easter, to Lower Edmonton. There lives my wife and my wife's, yes, my wife's, my wife's sister; he is a cabinet maker at Easter, at Easter" . . were constantly heard.

He signed his name with great satisfaction; the signature, however, was somewhat illegible and tremulous. In writing his address he misspelt words and, after looking at what he had written, rewrote some of the words which he had spelt wrongly or attempted to polish up the letters. To dictation he wrote single words of several syllables correctly, but was unable to take down correctly a sentence containing more than about five words. After writing a complex sentence he would look at the result, and, becoming dissatisfied, frequently crossed out all that he had written, saying "No, that's not right; I can't do it, I'm losing my senses." In writing to dictation perseveration frequently occurred; for example, he wrote "The London hospital hoptal is the the largst largest larst hostal in London."

He enjoyed reading the newspaper to himself, but easily tired. After reading for four or five minutes he would throw the paper aside to take it up again a few minutes later. He seemed to understand what he had read and would attempt in broken phraseology to retail the occurrences of the War, of which he

had just been reading. When asked to read aloud he managed simple words and sentences fairly well, and understood what he had read; long names and unusual words, however, were bungled and incompletely articulated. Frequently too, he mixed up words from different lines of print, making the whole seem an unintelligible muddle.

After considerable hesitation he copied slowly written manuscript with a fair degree of accuracy, but when copying printed matter he made more frequent errors.

He repeated the alphabet without mistake, and could count readily up to a thousand. He knew all the multiplication tables. He found difficulty in calculating. In attempting to deduce his age from the date of his birth he said: "Let me see, I was born in seventy, seventy, seventy-three, yes, that would be eighteen seventy-three when I was born, so that my age, my age, must be. No, I can't do it, I'm sorry, I can't." When asked immediately after this the age of his child born in 1904, he answered without delay "seventy-three," then he paused a moment and said "I have two children, a boy and a girl, I will give you their ages in a minute, let me see, let me see, no I can't do it, no, no," but never found the answer. When asked if one is not 10 years old, he nodded affirmatively and said "Yes, that's right." The question, How old is your wife? was then asked. "My wife will be ten years old in November; no, that's wrong, my wife will be, will be, eleven years old come June" (his wife was born in June, 1874), "oh, dear, oh dear, I think I'm mad."

In answer to the query, How many are nine times eight? he said: "Let me see 3×8 are 24, 4×8 are 32, 5×8 are 40, 6×8 are 48, 7×8 are 56, 8×8 are 64, and 9×8 are 72: yes nine times eight are seventy-two."

On again being asked, How many are nine times eight? he again repeated the eight times table and arrived at the answer seventy-two. On being asked a third time, he recognized that the question was the same, and remarked, "How foolish, how foolish!" and again repeated the table and found the correct answer. For a few minutes then no words were spoken and he was heard to repeat under his breath, the eight times table through, completely twice, as far as nine times eight. He was then asked, How many are eight times nine? To this he answered, "Eight times nine are the same as nine times eight, but I can't remember how many that made."

Without paper he was able to add eight to twenty-seven and readily answered thirty-five, but when asked to add three hundred and twenty-seven and eighteen, he said "That's more difficult, let me see, eight and seven are, no, let me see, oh I can't do it, no, give me the paper," whereupon he wrote $\frac{\text{"327"}}{18}$ and then said "No, it's too hard," and gave up the attempt. He subtracted in his head eight from twenty-seven and gave the answer nineteen, but when given paper and having written down $\frac{\text{"327"}}{18}$ could not do the subtraction.

When objects were held in front of him, he preferred to take them in his left hand, but when his attention was directed to his right hand he would take them in this. He could imitate, with either hand or with both, movements made in front of him; but when attempting to carry out a movement with his right hand, the left often moved correspondingly, and frequently he would repeat the movement he had just finished before he attempted to carry out the new movement which he wished to imitate.

To command he could snap his fingers, whistle, make pill-rolling movements, shake his head, nod, laugh, look angry, groan, cough, touch his nose, put out his tongue, show his teeth, take a deep breath, &c. He could also make any movement of expression, beckon, salute, throw a kiss, clasp his hands, make a fist, prepare for fight, &c.

On request he could make simple movements for definite objects without having the object in his hand. He showed well how to knock at a door, count out money, draw a cork, turn a barrel organ, strike a match, with either hand, but in all these actions the movements performed with the left hand were better executed than those with the right; when using the right hand, more joints of the limb and frequently the whole of that half of the body partook.

With more complex orders the results were always less well performed than with simple orders. For instance, when told to put his left hand on his head, the movement was well carried out; but when told to put his left hand on his right ear, he first put his left hand to his left ear, and then put his right hand on his right ear; recognizing, however, that he had not done what he intended, he shook his head, looked around for a moment when a gleam lighted his face and he put his right hand to his right ear and his left hand to his left ear. He was then told to put his left forefinger on his nose, whereupon he put his left hand on his left ear and then placed his right forefinger to the bridge of his nose. At the question, Is that right? he put his left forefinger on to his nose, and then quickly put his right hand to his right ear saying, "Now that's it, isn't it?"

On being asked next to make a fist with the left hand, he made a fist with both hands. He was then told to open his left hand, whereupon he opened his left hand, then half closed it, opened his right hand, half closed this, and finally opened both hands.

When asked to make movements as of cutting out with scissors, he made with both hands irregular movements for clumsily using scissors; a moment later, after gazing at his hands, he stopped the movements with his left hand whilst continuing those with the right.

He was able to make any simple movements on request; he touched his ear, scratched the middle of his back, pulled his hair, touched his great toe, tickled himself on command. He was equally certain when asked to touch similar parts of the observer's body. With objects in his hands he could brush his nails, comb his hair, blow his nose, &c. When given a paper knife and asked to show how this should be used, he folded the bed-clothes and then flourished the knife as if it were a butcher's. When asked to indicate

the use of a pencil he wrote his name on paper; and when given a pair of scissors and asked to show how they should be used, he took them in his right hand and began to perform cutting movements clumsily, but, becoming dissatisfied, asked for a piece of paper saying, "I can't use these things without a something to cut."

When asked to show how to thread a needle, no needle being given, he carried out the necessary movements more successfully than when he actually held the needle and thread. When shown a key, and asked to indicate how this should be used, he went through the movements of inserting the key and unlocking a door in a more or less complete fashion. He was then given a closed padlock and key, and asked to use the key, whereupon, holding the lock in his right hand he fumbled with the key; eventually he succeeded in inserting the key into the hole, but even then was unable to unfasten the lock. When given a comb, and asked to show how it should be used, he clasped the comb tightly in the palm of his right hand, passed it into the palm of his left, worked it up to his fingers and immediately started to comb his hair by moving his head against the comb.

A lighted match was placed between the fingers and thumb of his right hand; as the match burnt he skilfully worked it up his fingers, and when nearly burnt blew out the flame.

When given a box of matches and a pipe loaded with tobacco, he placed the pipe in his mouth, held the box of matches clumsily in his right palm, opened the box with his left hand, took out three matches, discarded two of these, closed the box and then made several attempts to strike the box on the match; after several such movements the match lighted, whereupon he dropped the match box, transferred the lighted match to his right hand and carried it to his pipe, but in puffing at the pipe blew out the light. He then repeated the performance but could not light the pipe. After two attempts he remarked, "No, no, I can't do it, though I should like a smoke." When given the match already lighted he succeeded in lighting his pipe at the first attempt, and when asked to strike a match he did so readily.

When given a piece of string, and asked to tie a knot on it, he held the string tightly between the clenched fingers and palm of his left hand and attempted with his right hand to throw a loop and catch the string with his left index finger. In this manœuvre he made many attempts, but in each failed; eventually after trying five minutes he gave up, saying, "I know how to do it, I've tied so many in my time, I've been in so many kinds of business and in all I had to tie knots, but, you know, all my skill for such things has gone. Almost everything with my fingers I've lost most." At the same time he could distinguish and name most types of knots, e.g., sailor's, granny, reef, &c.

When given five pennies and a halfpenny and asked to count the money, he placed the coins in his left palm and, touching the pennies in turn with his right index finger, counted "One, two, three, four, five, five pence halfpenny, that's right, isn't it?" On being given two florins, a shilling and sixpence

he counted the money from his left hand into his right remarking, "Two, and two, and one, that is four, and three, and six, no, no, that is four and sixpence, that is five and sixpence, yes, yes, that is five and sixpence. I know that, that (the florin) is two shillings, and that, that is twice that (the shilling) and that is a sixpence." Given gold, silver and copper to the value of £3 4s. 11½d., he made several attempts to count it out, but could not give any result; when eventually told the amount he remarked, "Yes, yes, I know how much it is, you can't do me! but I can't tell you."

Five plates were placed on a table in front of the patient, and were audibly and slowly numbered 1, 2, 3, 4, 5, the exact plate being indicated with the finger as the number was called; on the table to the right of the plates were placed a penny, a sixpence, a florin, a half-crown, and a sovereign. He was then asked—

To put the sixpence on plate 5.	Performed.
To put the sovereign on plate 1.	At this he put the sovereign on plate 5, waited a moment, and then took it out and placed it on plate 1.
To put the half-crown on plate 1.	Performed.
To put the florin on plate 4.	Whereupon he put the florin on plate 1; when asked if this was right, he took sovereign from plate 1 and placed it on plate 4.

When the test was made more complicated he usually paused and carried out a part of the command; on other occasions he would not attempt to perform any of the movements, although apparently he had understood, for he would remark, "I know what you say, and what I would like to do, but my hands seem all wrong." With practice on any one day the results obtained with this mode of testing became more and more accurate, provided that the orders were not made too complex, but on different days the accuracy of the results varied greatly. More accurate results were always obtained if the number given to the plates in front of him was written on them, provided that the numbers ran consecutively; if, on the other hand, the numbers on the plates were in the order, say, 5, 3, 4, 1, 2, the patient rarely succeeded in placing three of the coins consecutively on the desired plate.

When shown objects and asked to name them, he said when shown—

A pin.	(Readily) "pin."
A needle.	(Pause) "pin."
A pencil.	(Longer pause) "pin."
A watch.	"Half-past nine" (correct).
Yes, but what is it?	"Time piece."
Watch pocket in vest.	"Vest pocket."
The watch again.	(Readily) "vest-pocket holder"; he then became angry, shook his head and began to weep.

A medicine bottle.	"Bottle."
What does it contain?	"My medicine."
A bunch of grapes.	(Long pause and then) "rubber balls." After eating one he remarked, "I was driven to say rubber balls, oh, I know what they are; they are rubber balls and very good at that: yes, yes, very good rubber balls."
But they are not made of india-rubber, are they?	"I know that well enough, but words won't come, I can't think what they are."
On being told that they were grapes his face beamed, he nodded and said—	"And very nice, too."
A piece of string.	"String."
An electric light bulb.	"Electric light bulb; yes, we make those at our works." (Words badly articulated.)
A nail-brush.	"Oval brush"; then he began to show how it should be used; as soon as the brush touched his finger nails, he remarked, "no, a nail-brush."
The watch again.	(Readily) "time piece."
A towel.	(Pause, ten seconds) "towel," and then he read off the stamp, "London Hospital, Charrington Ward."
An inkstand.	(Quickly) "inkstand."
A pocket-knife.	"Knife, yes, pocket-knife."
A fountain pen.	"Penholder."
A pencil.	(Pause) "pen, no, fountain pin."
A pin.	(Instantly) "pin."

When a pin was placed in his right hand and a needle was put into his left, and he was asked, What is the difference between a pin and a needle? he remarked "The two extremities of the one are sharp, the pin is sharp, one end is sharp, the other end is sharp, is blunt." The needle was then removed from his left hand, leaving the pin in his right, whereupon, pointing to the blunt end of the pin, he said, "Now that end is both sharp, one end I can put through it, it has got a sharp extremity, the other"—continuing to fumble with the pin—"now that end is both sharp, let me see, let me see, I know, no, I'm sorry, but I can't tell you." Given the needle into his left hand, whilst the observer held up the pin in front of him, he said, "That's better!" pointing to the point of the pin with his right hand, "That's a plain point," and pointing to the head of the pin, "That's dull and has no point at all." Then examining the needle in his left hand and pointing to the eye of the needle, "That has got a needle and a cotton goes through it, a needle has a

cotton to it." He was then given the pin and a length of cotton thread, whereupon he spent two minutes vainly attempting to pass the thread through the head of the pin, fumbling in a clumsy fashion and occasionally putting his left hand to his head as if in thought. At the end of this time he remarked, "No, no, let me see, let me see." On being given the needle and the thread, he said, "Now we're off," and spent two minutes more in vainly attempting to thread the needle, eventually giving up the attempt, saying: "My right hand is a fool, I can't use it at all."

The number of times the patient repeated any given action bore little or no relation to the accuracy of the performance of that action. Moreover, no improvement in the mode of carrying out the action could be obtained after showing him how to use objects placed in his hands. A movement once initiated, either voluntarily or in response to a command, could be continued after the closure of his eyes. Even with his eyes closed he was able to place his right upper extremity into any position into which his left had been placed either actively or passively. No differences between the accuracy of imitation of postures from right to left and from left to right could be determined. The hands partook normally in general movements of the body.

His ideas of objects seemed to be normal; he had knowledge of the desired results of actions which he wished to perform, of the parts of the body which he would have to move, and of the order of their movement in order to accomplish the desired action.

The general condition of the patient altered little during the first three weeks after his admission, but the interferences with speech, with the performance of purposive movements, and with memory varied considerably from day to day and from moment to moment. When the pains in his head were severe, his memory was less certain, and his ability in carrying out movements with objects and on command diminished. From examination to examination and during each examination the results obtained in answer to tests varied greatly, although the type of intellectual loss remained constant; the variations observed seemed to be determined by the amount of attention the patient was able to concentrate on the required thought or action; at times little or no loss appeared, at others the loss was great.

No actual loss of power and no definite paresis of any kind was discovered until September 5. On the night of the 4th to 5th the power in his left upper extremity diminished to such an extent that the grip on this side, measured on the dynamometer scale, fell in twenty-four hours from 80 to 30, whilst that of the right hand remained unchanged between 70 and 80. After this date he became more drowsy and heavy, and to testing less certain in his answers; at the same time the headache became more constant and more severe.

A radiographic negative of the skull taken on September 3 shows a loss of definition of the upper margins of the sella turcica, but no changes in the outlines of its floor or elsewhere in the skull, and no alteration in the density of the soft tissues of the cranium.

Gradually the weakness of the left upper extremity, first noticed on September 5, extended. On September 24 the patient was characteristically hemiplegic. The muscles of the left forearm and hand were absolutely flaccid and the patient was unable to move this extremity. He could not lift his left leg from the bed, but was just able to flex the knee and dorsiflex the ankle. The left leg was somewhat hypertonic and stiff. On the left side the knee-jerk and ankle-jerk, and to a less extent the deep reflexes of the arm, were exaggerated; ankle-clonus could not be obtained, but *both* plantar reflexes gave an extensor response. He could no longer control his sphincters.

Under observation the signs of disease in the chest gradually extended and before death affected the whole of the left lung; at the same time catarrhal signs appeared in the right lung. On September 22 the temperature became raised and irregular, and the rate of the pulse rose to 120 beats per minute; on the 28th the temperature reached 104° F., the pulse was uncountable at the wrist and the rate of respiration was 50 per minute. On the morning of the 29th, forty-four days after admission and eighty days after the onset of the squint, he died. An autopsy was performed on October 1.

AUTOPSY, OCTOBER 1, 1914.

Occupying the lower and posterior half of the lower lobe of the left lung was an ill-defined mass of carcinoma, measuring 6 cm. in diameter; the apex of the lower lobe also showed carcinomatous infiltration. A small secondary carcinomatous nodule was found in the centre of the lower lobe of the right lung. Secondary nodules of carcinoma, up to 3 cm. in diameter, were present in the glands at the hilum of the left lung and at the bifurcation of the

trachea; at the hilum of the right lung smaller secondaries were seen. The left lower paratracheal glands contained masses of secondary growth 2 cm. in diameter, and the right a mass measuring 4 by 2·5 cm., which was continuous with another mass, 7 by 4 cm., in the superior mediastinum. The left pulmonary vessels were constricted by these secondary glands. The lower end of the trachea and the commencement of the left bronchus were infiltrated by carcinoma, which had spread from the paratracheal glands. Masses of secondary carcinoma were also found in the right and left lower cervical glands, in the glands of the lower portion of the mediastinum and in the pancreatic glands. A secondary nodule measuring 6 by 5 cm. was found in the right suprarenal gland and a similar mass, 3 by 3·5 cm., was found in the left.

The brain weighed 1,439 grm. The meninges appeared normal. All the convolutions were flattened. The left supramarginal convolution contained a nodule of secondary carcinoma 2·5 cm. in diameter. This nodule was slightly adherent to the meninges, had a rounded form, and extended deeply into the subcortex. It was surrounded on its upper and anterior border by recent anæmic softening. The lower half of the post-central convolution was diffluent. The superior temporal, the angular, the upper part of the post-central, and the superior parietal gyri were unaffected, and no gross changes were seen in the other convolutions of the hemispheres. In macroscopical section the corpus callosum, the tapetum, and the inferior longitudinal bundle appeared normal.

The whole of the pituitary gland and the meninges around it were infiltrated with carcinomatous growth, which had eroded the floor of the sella turcica, and invaded, as a mass the size of a marble, the sphenoidal fissure.

In the medulla of the right femur, at the junction of its middle and lower thirds, an osteoplastic secondary nodule of carcinoma 1·3 cm. in diameter was discovered.

The upper part of the upper lobe of the left lung was collapsed and œdematous, whilst the lower lobe on the right side showed bronchopneumonic consolidation and mucopurulent bronchitis. Over both lungs a certain amount of serofibrinous pleurisy was discovered. The heart appeared normal; the liver and kidneys were congested, and the spleen was slightly enlarged and septic (weight 241 grm.).

Microscopic Examination.—Portions of (1) the left lung, (2) a lower cervical gland, (3) the left suprarenal, (4) the pituitary gland, and (5) the nodule in the left supramarginal convolution of the brain were embedded in paraffin; sections were stained by hæmatoxylin-eosin and van Gieson. The carcinoma is similar in appearance in all sections. It consists of tubules lined by columnar, cubical or polygonal cells, and less numerous, solid processes composed of polygonal cells. A little secretion is present in some tubules. There are areas of necrosis, which are infiltrated with numerous neutrophile leucocytes.

The pituitary gland is replaced by growth, with the exception of a narrow semilunar portion of the anterior lobe; in this remnant of the anterior lobe there are patches of necrosis.

The section of the brain includes half the rounded carcinomatous nodule and the convolution anterior thereto. The upper surface of the growth reaches the leptomeninges. The growth is confined to the supramarginal convolution; it is separated from the adjacent post-central convolution, in its upper part by a narrow strip of degenerate cortex, in its lower part only by leptomeninges in the intervening sulcus. The post-central convolution is œdematous, and many of its nerve-cells show considerable evidence of degeneration, for instance loss of angularity. The cortex opposite the lower part of the growth shows severe degeneration. In the anterior convolution there are no fat-granule cells.

For the notes on the autopsy I am indebted to Dr. Hubert Turnbull, Director of the Pathological Institute; my best thanks are also due to Dr. Lewis Smith under whose care the patient was admitted.

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A HITHERTO UNDIFFERENTIATED NUCLEUS IN THE FOREBRAIN (*NUCLEUS SUBPUTAMINALIS*).[1]

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In this short communication it is desired to call attention to a somewhat important group of nerve-cells which have not been identified with any anatomical formation hitherto described. This formation was observed while studying the structure of the basal ganglia, especially the nucleus lenticularis, in the human brain and that of the monkey, by means of horizontal and frontal serial sections, stained by Nissl's method. Especial attention was paid to the surrounding structures connected more or less intimately with the nucleus itself. The most difficult of these formations to study, on account of the variability and irregularity of its arrangement as regards the nucleus lenticularis, is the substantia perforata anterior or lobus parolfactorius (Edinger). In this region, according to the work of Beccari [2], in all mammals, including man, we must distinguish: (*a*) a small anterior zone (eminentia parolfactoria), not always apparent in man, situated immediately behind the trigonum olfactorium; and (*b*) a large zona postero-lateralis (planum septale) in which there can be distinguished a more or less evident ridge, corresponding roughly to the diagonal bandlet of Broca, which is not always present in the human brain.

While studying the structure of the different formations of the substantia perforata anterior in man in relation to the overlying ganglion mass of the nucleus lenticularis, a group of nerve-cells of considerable size was observed which I shall call *nucleus hypolenticularis*, or more exactly, *nucleus subputaminalis*. An attempt to justify this appellation is given below.

In order to make clear the topographical position of this nucleus, it is well to remember some details of the external conformation of the ventral face of the nucleus lenticularis, and the various relations which

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this face has to the underlying structures. As is known, the ventral face of the nucleus lenticularis, at that point where the antero-ventral part of the putamen joins the head of the nucleus caudatus is crossed by a channel in which runs the lateral portion of the anterior commissure. This last, following a curved course in an oblique direction from interior to exterior, from front to back, and from above downwards, is submerged in the substantia perforata anterior and enters the above-mentioned channel. On this account the channel was called the canal of the commissura anterior by Gratiolet. Anteriorly the channel is situated between the ventral face of the head of the nucleus caudatus and the deep stratum of the pars anterior of the substantia perforata anterior, or more exactly, eminentia parolfactoria, in those brains in which the structure exists. The canal continues its course between the deep stratum of the planum septale and the ventral face of the nucleus lenticularis (which face is now formed by the putamen and the external segment of the globus pallidus), in such a way that the two segments of the nucleus lenticularis (ventrally) are separated from each other by the anterior commissure. Finally the commissure, crossing the inferior margin of the putamen at an acute angle reaches with its posterior extremity the lowest part of the external capsule and crosses the fibres of the fasciculus longitudinalis inferior. For this reason the ventral margin of the putamen is cut into two parts, one anterior, the other posterior. This is well shown in horizontal sections (somewhat obliquely inclined backwards) throughout the length of the lateral part of the anterior commissure. The anterior part, united with the head of the nucleus caudatus, extends distally like a beak external to the commissura anterior and internal to the lowest part of the capsula externa. The posterior part, on the contrary, remains interior to the commissura anterior, and exterior to the substantia perforata anterior and the substantia innominata of Reichert.

If we study the connexions of the ventral face of the nucleus lenticularis in vertico-transversal serial sections, it is seen that they change as we go from front to back. In fact, in front, in that plane in which the nucleus lenticularis is represented only by the anterior portion of the putamen, closely connected with the head of the nucleus caudatus, the ventral face of the nucleus lenticularis lies on the eminentia parolfactoria, and almost reaches the surface of the brain. More distally, on the contrary, that is to say as soon as we reach the plane in which the external segment of the globus pallidus appears, the ventral face of the nucleus lenticularis corresponds to the planum septale, from which it

is incompletely separated by the fibres of the commissura anterior and those of the ansa lenticularis.

We now note the appearance of the nucleus subputaminalis in the vertico-transverse and horizontal sections stained by Nissl's method. It is to be remembered that these sections must be cut strictly serially, i.e., it is necessary to stain all the sections of the series, otherwise the group of cells below described will be overlooked.

The nucleus subputaminalis begins to appear in the section passing through the level of the median part of the commissura anterior, namely, where the nucleus lenticularis is formed by the putamen and the external segment of the globus pallidus. Here the putamen is not well marked off ventrally from the deep stratum of the substantia perforata anterior (planum septale) and from the ventro-medial expansion of the claustrum. In this complicated and poorly determined zone (fig. 1) appears a small group of large nerve-cells, staining deeply with toluidin blue. These cells are elongated in shape. In the transversal direction, for the distance of about 1 mm., the group is in contact dorsally with the ventral margin of the putamen, the grey matter of which partly surrounds the medial apex of the group. In the photomicrograph the group of cells appears interrupted by a small blood-vessel.

Slightly distally, in the transverse section, at the level of the anterior part of the tuber cinereum, the ventral face of the putamen is still not well distinguished from the ventro-medial prolongation of the claustrum and from the grey mass which is below and behind, continuous with the nucleus amygdalæ. Here the cells of the nucleus subputaminalis appear slightly displaced to the outer side, in comparison with the previously mentioned section, and are larger in the transversal sense. It remains transversely in contact with the ventral margin of the putamen, except for a few fibres which probably belong to the capsula externa. The nucleus subputaminalis at this level (fig. 2) is of irregular shape and composed of a principal group of nerve-cells (fig. 2, a) and a small lateral group (fig. 2, b) united to the former by a few cells which are morphologically identical with those of the two groups. In all, the nucleus subputaminalis extends for 2 or 3 mm. transversely and for about 0·5 mm. vertically. All the cells composing the group are large and closely approximated (more so than in the preceding section) and in all cases stain deeply with toluidin blue.

In order to make the morphology and topography of the nucleus clear, a semi-schematic figure is given of a frontal section cut in a plane somewhat posterior to the one above described, i.e., at the level of the

infundibulum[7] (fig. 3). The ventral face of the putamen is well separated in this section from the claustrum and substantia perforata anterior, by the lateral part of the commissura anterior. Externally

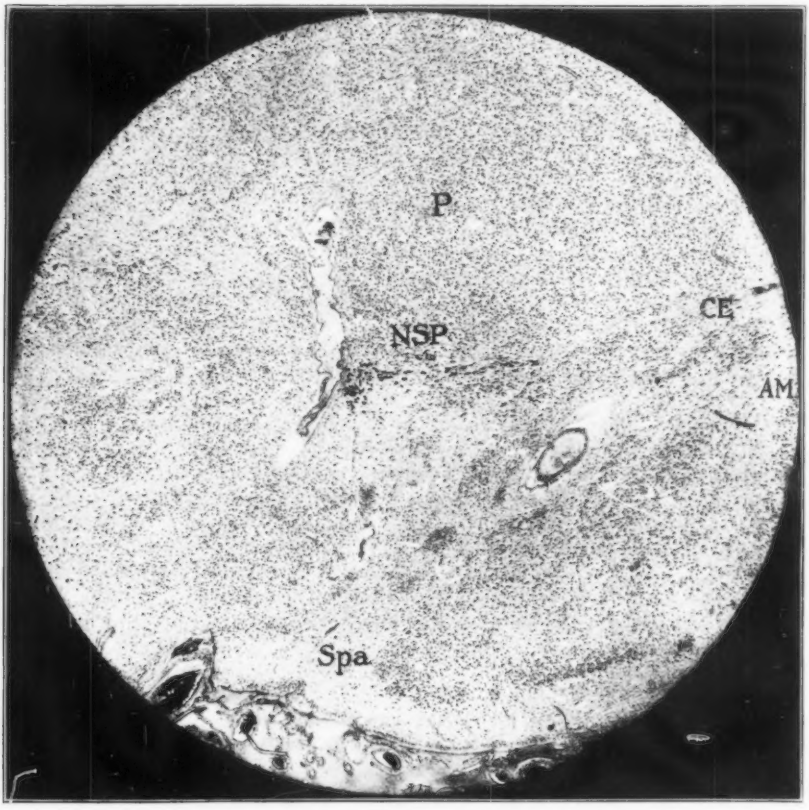

FIG. 1.—Photomicrograph from a frontal section of the right hemisphere at the level of the portio media of the commissura anterior. The proximal part of the nucleus subputaminalis is seen. AM — claustrum; CE — capsula externa; P — putamen; Spa — substantia perforata anterior.

to the commissura anterior, the nucleus subputaminalis is seen. It consists of large cells and extends transversely 3 or 4 mm. immediately below the ventral margin of the putamen. At this point the nucleus [seen better in the photomicrograph (fig. 4) than in the diagram] is

composed of a considerable group of cells lying transversely and extending medially as a prolongation, which, going upward and internally, enters into the angle formed by the ventral margin of the putamen and the

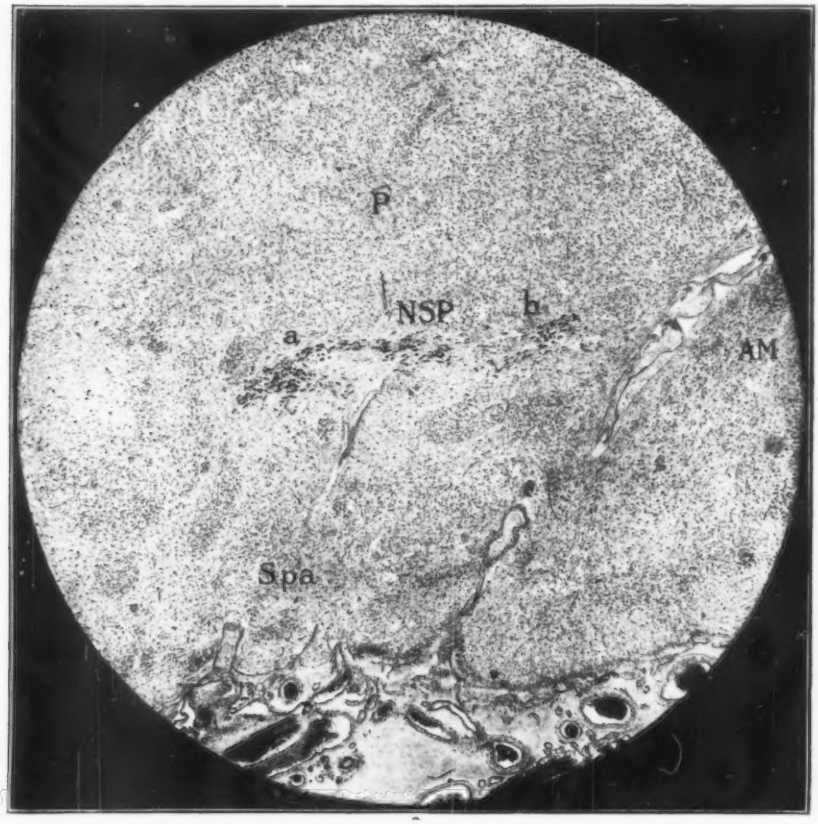

FIG. 2.—Photomicrograph from a frontal section of the right hemisphere at the level of the pars anterior of the tuber cinereum. The nucleus subputaminalis (NSP) is formed by two groups of cells—one medial (a), and the other lateral (b). Other lettering as in fig. 1.

external margin of the anterior commissura. Internal to the commissure, in the deep stratum of the substantia perforata anterior (planum septale) we can see another group of cells of medium size and sparse arrangement. This group extends laterally between the ventral part

of the globus pallidus and the commissura anterior, and is nothing else than the ganglion of the ansa peduncularis (Meynert [5]); nucleus basalis of Meynert (Kölliker [4]); nucleus of the planum septale (Beccari [2]); or ganglion of the ansa lenticularis (Edinger [3]).

In more distal frontal sections, the nucleus subputaminalis assumes a more lateral position and remains external and posterior to the external and posterior limits of the substantia perforata anterior. In the section immediately anterior to the corpus mammillare [fig. 5], we see that only the medial extremity of the nucleus subputaminalis remains. This extremity continues upward and inward with the prolongation, as in the preceding section. From its ventro-medial

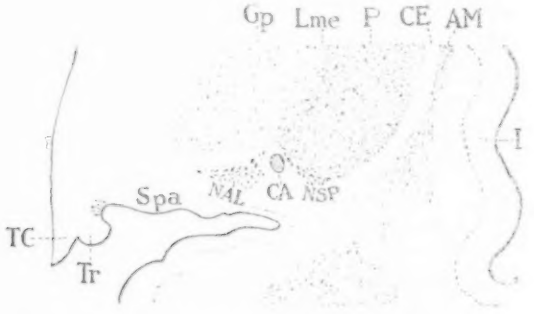

FIG. 3.—Semischematic figure (1 x 1) of a frontal section at the level of the tuber cinereum. Nissl stain—AM = claustrum; CA = commissura anterior; CE = capsula externa; Gp = globus pallidus; I = insula; Lme = lamina medullaris externa nuclei lentiformis; NAL = nucleus ansa lenticularis; NSP = nucleus subputaminalis; P = putamen; Spa = substantia perforata anterior; TC = tuber cinereum; Tr = tractus.

part some nerve-cells extend toward the nucleus ansa lenticularis. This last, however, remains internal to and below the commissura anterior and completely separated from the nucleus subputaminalis. As we gradually proceed distally, the group of cells, representing the medial extremity of the nucleus subputaminalis and its medial prolongation, gradually disappears.

In order to understand better the topography of the nucleus, it is necessary to study horizontal sections cut parallel to the orbital face of the lobus frontalis and, consequently, to the substantia perforata anterior. It is evident that, as shown by the frontal sections just discussed, the nucleus subputaminalis will not appear until we reach a section passing exactly through the level of the ventral face of the globus pallidus, i.e.,

a section in which, of the nucleus lenticularis, there exists only the ventral portion of the putamen, united anteriorly with the ventral portion of the nucleus caudatus. Proceeding from above downwards in the horizontal sections, the first appearance of the nucleus subputaminalis is the distal extremity which is a group of large cells situated exterior to the surface of the section of the lateral portion of the commissura anterior and internal to the ventral margin of the putamen. Distally this group of cells sends a prolongation back of the commissura anterior, thus coming in contact with the cells of the nucleus ansa lenticularis. This last remains entirely internal to the commissura, situated in the deep stratum of the planum septale. Although in the Nissl preparations of this level it is not possible to draw a sharp line between the nuclei, nevertheless the arrangement and morphology of the cellular elements indicate clearly enough where the one ends and the other begins. Certainly the horizontal sections confirm what we have seen in the frontal sections, i.e., that distally the nucleus subputaminalis is continuous with the nucleus ansa lenticularis by means of little branches of cells passing dorsally to the commissura anterior.

In a more ventral horizontal section (fig. 6) we see the ventral part of the putamen divided into a proximal and distal segment by the external striate vessels cut transversely. The internal margin of the proximal part is continuous with the posterior margin of the head of the nucleus caudatus, so that together they describe a curve with an interior concavity. In this concavity the commissura anterior and the nucleus subputaminalis are situated. The latter remains, throughout almost all its length, in contact with the median margin of the putamen; not all its length, however, for its distal portion is not seen because it lies a few microns dorsally (seen in the section last described). Here also the visible part of the nucleus subputaminalis describes a curve with the concavity inward. Antero-posteriorly it extends proximally to the point at which the putamen joins the head of the nucleus caudatus, and distally as far as the lateral margin of the surface of the commissura anterior, or more exactly, as far as the portion of the commissure which crosses the inferior margin of the putamen. Interior to the nucleus subputaminalis we see another group of small cells which may be identified with a fragment of the ventral portion of the putamen. The fragment lies in this situation on account of the direction which the commissura anterior takes at this point, where it crosses the above-mentioned channel. Thus the nucleus subputaminalis appears delimited laterally and frontally by the internal margin of the

putamen, and medially by a fragment of the putamen and commissura anterior which separate it from the nucleus ansa lenticularis. The details and arrangement of the nucleus are more evident in the photomicrograph (fig. 7), corresponding to the semi-schematic fig. 6. In

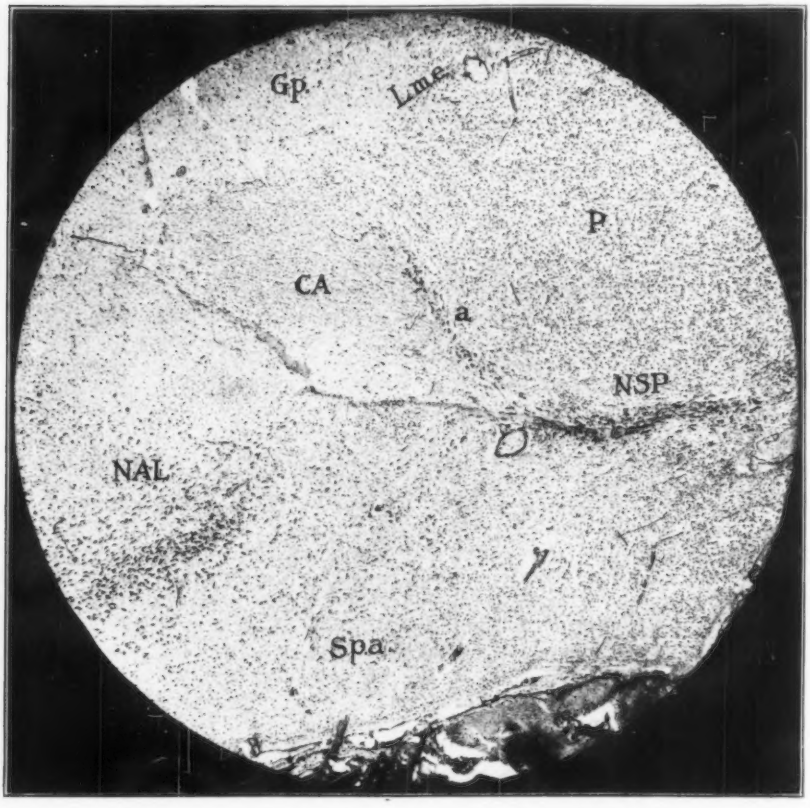

FIG. 4.—Photomicrograph from frontal section of right hemisphere at the level of the infundibulum. Medially from the nucleus subputaminalis a prolongation (a) is directed upward and medially between the putamen (P) and the commissura anterior (CA). Other lettering as in fig. 3.

the photomicrograph the nucleus subputaminalis appears of very irregular shape, inasmuch as the antero-median extremity is circumscribed, while in the median portion it becomes larger, then smaller and finally large again, till it reaches the vasa striata, which interrupt

it abruptly. Interior to the nucleus and in front of the superficial section of the commissura anterior, we see the fragment of the ventral portion of the putamen. Still more interiorly, as regards this last fragment and the commissura anterior, we can see some sparsely

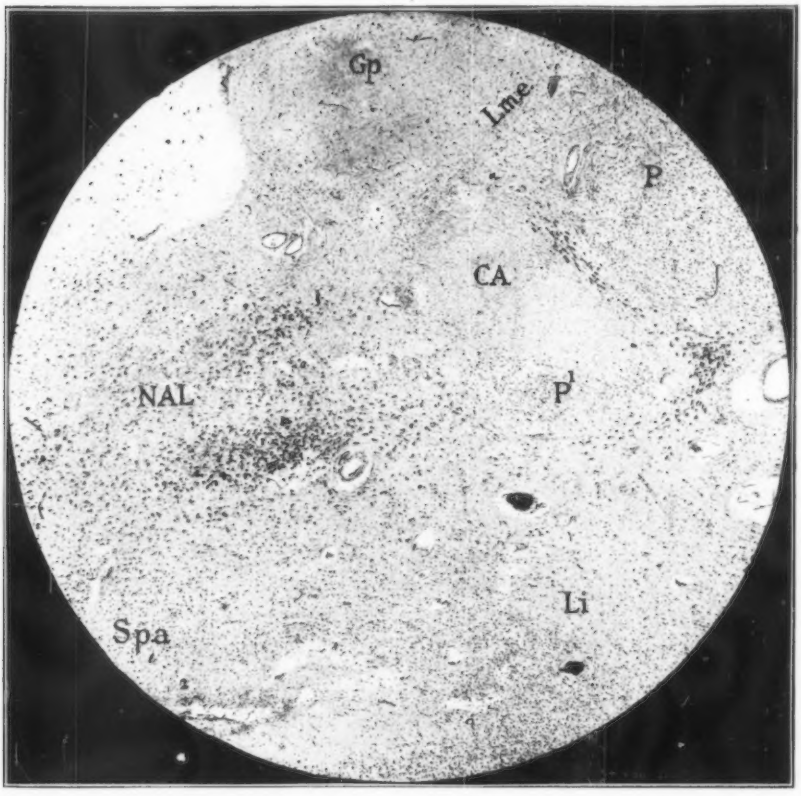

FIG. 5.—Photomicrograph of frontal section of right hemisphere immediately in front of the tuberculi mammillari. The nucleus ansa lenticularis (NAL) is enlarged, and remains clearly separated from the nucleus subputaminalis (NSP) by the commissura anterior (CA) and a fragment of the putamen (P'). The nucleus subputaminalis is reduced to a group of cells situated outside the substantia perforata anterior (Spa), corresponding to the limen insulae (Li). Other lettering as in fig. 3.

arranged nerve-cells. These are nothing else than the lateral portion of the nucleus ansa lenticularis. Anteriorly and exterior to the nucleus subputaminalis, we see the ganglion mass belonging to the

proximal part of the ventral margin of the putamen. The same photomicrograph shows the disposition of the cells of the various segments of the nucleus subputaminalis. Here, although irregularly distributed, the cells are in general closely approximated, and it is ot possible to differentiate the individual cells with the low power objective. The cells of the nucleus ansa lenticularis, as has been mentioned, are, on the contrary, in more or less dense groups which, although also irregular in arrangement, are always clearly separated from one another.

Proceeding vertically by means of horizontal sections, the nucleus subputaminalis disappears altogether.

At this point, it is best to explain that the appearance noticed in the above-mentioned frontal and horizontal sections is not constant, and in certain other sections assumes a different form. The reason for this variation is (1) because the nucleus does not lie exactly in a horizontal plane, and (2) because it describes a curve in the antero-posterior direction. Besides the disposition of the nucleus differs somewhat in the two hemispheres, for, adapting itself to the ventral margin of the putamen and following the individual variations in the form and direction of the latter, it necessarily varies with it. Nevertheless, in the horizontal sections the nucleus subputaminalis, on the whole, describes a curve with the concavity internal and posterior. It is larger in its median part than in the proximal and distal extremities, as shown in the vertical transverse sections.

Resuming the description of the horizontal and frontal sections, we can say that the nucleus subputaminalis is a dense group of cells situated beneath the ventral portion of the putamen (portio media), at the point where this margin is crossed by the commissura anterior. Although its contour is somewhat irregular, as a whole it is flat in the vertical and curved in the horizontal plane. The direction of its greater axis is oblique, passing from the exterior inward and from front to back. It extends antero-posteriorly for about 1 cm. from a frontal plane which passes through the median part of the commissura anterior to another frontal plane passing immediately in front of the corpus mammillare. Transversely it measures at its greatest length at least 2 or 3 mm. It is bounded (*a*) dorsally by the inferior margin of the putamen, from which it remains separated by some ventral fibres of the capsula externa; (*b*) internally, in part (distally), by the commissura anterior, and in part (proximally) by a fragment of the putamen which surrounds its medial extremity; (*c*) ventrally and externally by fibres of the capsula externa, and distally by the complex ganglion mass which constitutes the limen insulæ.

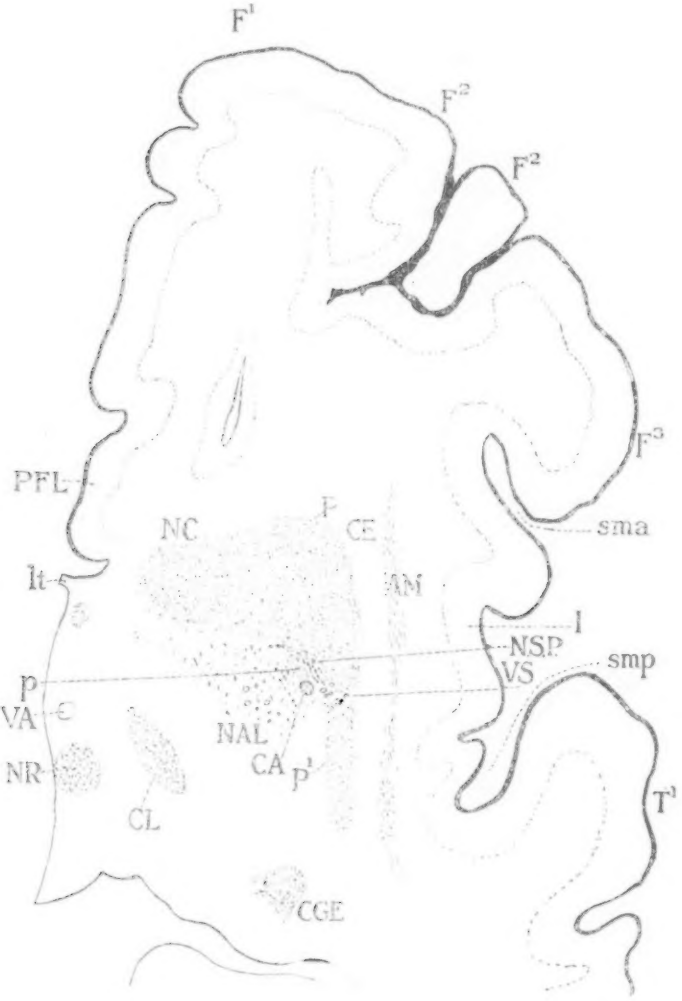

FIG. 6.—Semischematic figure (1 x 1) of a horizontal section parallel to the ventral face of the globus pallidus, immediately below the ansa lenticularis. CL = corpus luysii; CGL = corpus geniculatum lateralis; F^1 F^2 F^3 = gyri frontales; lt = lamina terminalis; NC = nucleus caudatus; NR = nucleus ruber; P = putamen (proximal extremity); P^1 = putamen (distal extremity); p = putamen (fragment of the ventral margin); PFL = area parolfactoria ("piega-fronto-olfatto-limbica"); sma = sulcus marginalis anter. insulæ; smp = sulcus marginalis poster. insulæ; T^1 = gyrus temporalis superior; VA = fasciculus thalamo-mammillaris; VS = arteriæ striatæ externæ. Other lettering as in fig. 3.

The cells of the nucleus are easily distinguished in the Nissl preparations from the adjoining grey formation, by their morphology and structure (fig. 8). The most prevalent forms are oval, pyriform and sometimes fusiform. They measure in greatest diameter 40 to 50 microns. Many appear in frontal sections with the greater axis transverse.

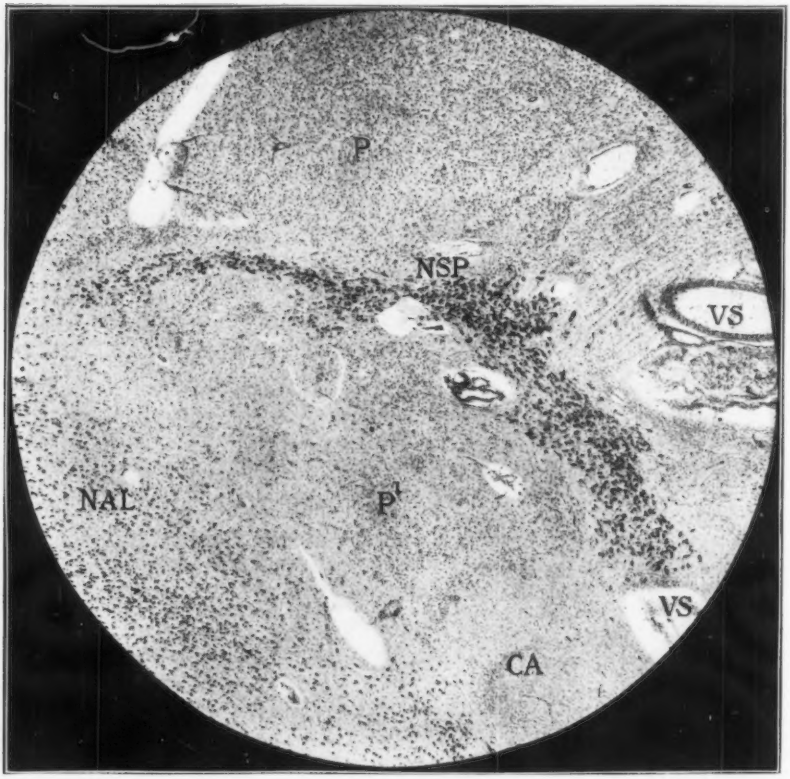

FIG. 7.—Photomicrograph of a horizontal section immediately below the ventral face of the globus pallidus. Lettering as in fig. 6.

The nucleus is of moderate size (12 to 14 microns), vesicular or ovoid in shape and somewhat poor in chromatin, and the situation is always excentric. There is a small, somewhat round, deeply staining nucleolus. The cytoplasm, which makes up a considerable part of the cellular body, is very rich in tigroid substance. This appears (*a*) in the form of large

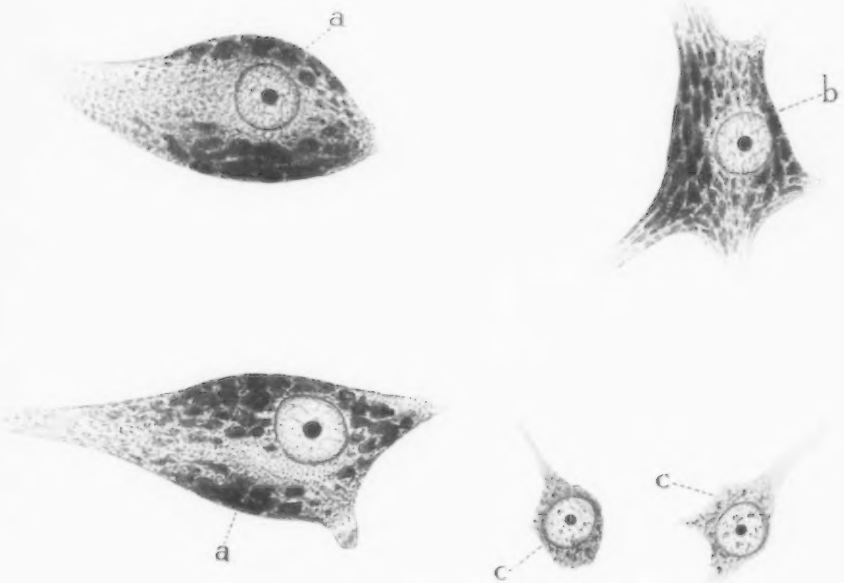

Fig. 8.

a = Nerve-cells of the nucleus subputaminalis.
b = Nerve-cell of the n. ansa lenticularis.
c = Nerve-cells (middle size) of the putamen.

Preparations from the brain of a patient dead of lobar pneumonia, fixed in alcohol and stained with toluidin blue. Drawn by artificial light. Objective $\frac{1}{12}$, ocular 6 (Leitz).

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somewhat round masses, staining deeply with toluidin, and which, although irregularly distributed, are dense at the periphery, or at the two poles of those cells which are of elongated form ; and (*b*) in the form of small granules staining more or less deeply and sparsely distributed throughout the cytoplasm. Both the masses and the granules of the tigroid substance are rarely continued into the dendrites, where they assume an elongated shape. A peculiarity of these cells is that they stain intensely with toluidin, thionin, methylene blue, &c., even in weak solutions. They retain their stain long after the rest of the section is almost discoloured by age.

It might be supposed that the nucleus above described represents a detached portion or appendix of some adjacent anatomical structure, from the fact that it is found near the putamen, the ventro-medial portion of the claustrum, the substantia perforata anterior, and the nucleus ansa lenticularis. This last is considered, by Beccari, a deep formation of the planum septale. The reasons for distinguishing the nucleus subputaminalis from the above structures are as follows. First of all, a glance at the sections or corresponding photomicrographs is sufficient to exclude the possibility of its belonging to the putamen or claustrum, partly because these two are clearly separated from it, and partly because the cells are absolutely different in size, form, and structure, from those of the nucleus subputaminalis. In fact the greater part of the cells of the putamen (shown in a former publication on the nucleus lenticularis [1], are small or of medium size (8 to 15 microns), inconstant in shape, and having a relatively large nucleus. The cells are poor in cytoplasm and chromatin granules. It is true that in the putamen there are some giant cells, but they are very rare, isolated, and well distinguished, on account of their size, polygonal shape and structure, from those of the nucleus subputaminalis. The cells of the claustrum, although a greater part are a little larger than those of the putamen, never reach the size of the cells of the nucleus subputaminalis. Their shape in this zone is, in general, fusiform. They are situated in the long axis vertical and almost parallel to the fibres of the capsula externa.

As regards the connexion of the nucleus subputaminalis with the substantia perforata anterior, it is first to be pointed out that the greater part of the nucleus subputaminalis is situated externally and behind the substantia perforata anterior, i.e., immediately above the point where this substance is continuous with the limen insulæ. Only a small part (proximally) corresponds to the deep stratum of the substantia perforata

anterior, or more exactly its external zone according to the division of Cajal [6], or to the planum septale, according to the division of Beccari [2]. Cajal, speaking of the external region of the substantia perforata anterior in man, describes four strata, three formed of cells and one of fibres. He does not mention a ganglion of large cells, situated and shaped as above described for the nucleus subputaminalis. In the deeper stratum of this region (stratum of fusiform and triangular cells) can be found in all cases, according to Cajal, certain other islets of cells widely separated from each other. These cells are giant, stellate, triangular, or fusiform in type. "They resemble," he says, "motor cells in shape and structure, as they possess an abundant protoplasm, rich in masses of chromatin and a great quantity of yellowish pigment." Evidently Cajal did not refer to a well defined formation, but rather to the cells of the nucleus ansa lenticularis which here and there appear irregularly grouped.

Even Beccari, in a most accurate description of the parolfactory region, describes in the planum septale (external region of the substantia perforata anterior of Cajal) two superficial strata of cells, and adds that the nucleus ansa lenticularis of Meynert, to which he gave the name of nucleus planum septale, is above them. He here follows the description of Kölliker. If this region is studied by means of Nissl's method of staining, it seems at first sight that the group of cells situated under the putamen is nothing more than the nucleus ansa lenticularis. However, if studied systematically in a complete series of horizontal and frontal serial sections, as pointed out above, one sees that it is a distinct formation, although distally connected with the nucleus ansa lenticularis. In fact the latter, as Kölliker and Beccari have shown, begins distally in the vicinity of the posterior extremity of the corpus mammillare, as a group of cells lying between the optic tract and the cauda of the commissura anterior, corresponding to the inferior extremity of the lamina medullaris externa of the nucleus lentiformis. This nucleus, covering the ventral face of the globus pallidus, extends frontally until some of its cells reach the posterior margin of the head of the nucleus caudatus. Although the nucleus ansa lenticularis extends, by means of prolongations, between the globus pallidus and the commissura anterior, there is no doubt that its body proper remains internal to the commissure, and below the ventral face of the globus pallidus, from which it is separated by means of the fibres of the ansa lenticularis. On the contrary, as we have seen, the nucleus subputaminalis is situated, as a whole, immediately external and slightly ventral to the commissura anterior (portio lateralis) and is approximated to and almost surrounded by the grey substance of the ventral margin of the putamen.

The reason for the distinction between this nucleus and that of the ansa lenticularis is that there exist certain structural and morphological differences between the elemental cells composing them. The cells of the nucleus ansa lenticularis are irregularly arranged and always widely separated from each other and form a more or less dense stratum which occupies the deep layer of the planum septale. On the other hand, the cells of the nucleus subputaminalis are densely and uniformly arranged and constitute a very compact group hardly distinguishable with the low-power objective. Still, the individual cells of the nucleus ansa lenticularis, although they are fairly large (20 to 30 microns according to Kölliker), and rich in chromatin, as are those of the nucleus subputaminalis (fig. 8*b*), in general, are of smaller size, since those of the latter measure 40 to 50 microns. While the shape of the cells of the nucleus ansa lenticularis is multipolar and polymorphous, that of the cells of the nucleus subputaminalis is elongated, oval, or pyriform. Finally, the structure of the two groups of cells appears somewhat different inasmuch as the tigroid substance of the cells of the nucleus subputaminalis occurs in the form of small granules or large masses disposed usually at the periphery of the cells, while in the cells of the nucleus ansa lenticularis the chromatin masses are smaller and irregularly distributed.

For the above reason it seems justifiable, from a descriptive point of view, to differentiate the one from the other, and to individualize the nucleus subputaminalis as a distinct anatomical formation. It is here intended by the term "nucleus subputaminalis" simply to indicate the topographical situation and to leave open the question of its morphological significance and functional value. Nevertheless, it seems highly probable that the nucleus subputaminalis and the nucleus ansa lenticularis, which might be called *nuclei hypolenticulares*, are, from the point of view of general morphology, formations which bear the same relation to each other as the putamen and nucleus caudatus.

The nucleus above described in man was found well developed in some monkeys in which, although smaller than in man, it preserves its characteristic position under the putamen, and is always distinct from the nucleus ansa lenticularis.

General Explanation of Figures.

The photomicrographs and semi-schematic figures are taken from frontal and horizontal serial sections, fixed in alcohol and stained with toluidin blue. The block, from which the sections are cut, includes all

the basal ganglia. The photomicrographs are all of the same enlargement and show the nucleus subputaminalis and the surrounding structures.

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Publications Recently Received.

[Notes on a book under this heading do not preclude a subsequent review.]

Die Lokalisation im Grosshirn. Von Professor C. v. Monakow S. 1033. Mit 268 Abbildungen im Text und 2 Tafeln. Wiesbaden: Bergmann, 1914.

Von Monakow has always been strongly opposed to the current views on cortical localization. In this book of over a thousand pages, he has considered every aspect of the question, summing up his ideas on the subject at the end of each section.

The key to von Monakow's position is an understanding of the process he has called "diaschisis." Although it is one of the accompaniments of surgical shock, the two conditions are in no sense the same; diaschisis is a cessation of function starting from the local lesion and extending to normal portions of the grey matter which receive fibres from the injured part. This is not primarily a loss of function following physiological lines, but is a nutritional change following a strictly anatomical distribution. The effect of the lesion "cleaves through" neuronal junctions, so that the loss of function attacks parts belonging to distant neuronal systems, which, however, are connected normally with the part affected. Thus, for instance, a cortical ablation may cause a loss of motor power corresponding to that due to lesions of the anterior horn-cells, or hemianalgesia resembling that seen in lesions of the mid-brain.

It is at once obvious that, on this view, the ordinary conceptions of cortical localization are erroneous, and the evidence on which it is based fallacious. Von Monakow, therefore, begins by considering how function returns after an experimental lesion, and points out the long duration of diaschisis. He then considers, one after the other, the various methods used to prove the existence of motor and sensory centres in the cortex.

These are (1) extirpation and examination of the subsequent loss of function; (2) physiological excitation; (3) anatomical methods, including the study of the cyto-architecture; (4) the method of comparative anatomy; and lastly (5) the clinical anatomical method.

The usual conception of a motor centre is a localized area from which single definite movements can be evoked, in some part of the opposite half of the body, by local stimulation; at the same time removal of this area should cause paralysis in that part. But, according to von Monakow, these "centres" are not the places where voluntary movements are manufactured, but nodal points for the production of synergic movement out of a host of motor impulses, called into activity for the most part reflexly. Destroy these points and the

only specific effect is the diminution of co-ordination and synergy, although the immediate consequence is the production of a gross paralysis by diaschisis. Thus he believes that all the physiological evidence deduced from ablation and stimulation is fallacious, provided it is used to prove the localization of the lost function or the movements following excitation in that part of the cortex.

He also believes with Brodman that cyto-architecture is no clue to function, instancing the absence of large pyramidal cells from that area of the cortex where movements can be excited in the arm. Moreover, he thinks that no anatomical motor area can be proved to exist even in the monkey by ablation of the cortex and subsequent study of degeneration of the pyramidal tracts.

He sums up his views on localization of movement on p. 215; there is, he believes, a cortical somatic zone, but this is simply the spot of most complete representation of movement ("Optimale anatomische Repräsentationsbezirke"). Any movement evoked by the will demands the activity of a host of other centres, not only lower in the nervous system, but also in other parts of the cortex. This simultaneous working of centres at various levels of the nervous system in many ways closely resembles the views of Hughlings Jackson, which seem to be unknown to von Monakow.

He then considers the localisation of sensibility, and here we think the German method of talking about "bewusste und unbewusste Sensibilität" for processes in the spinal cord and mid-brain militates against clearness of exposition and thought. Sensation is a mental process, and the English terms "sensory and non-sensory afferent impulses" would have rendered von Monakow's handling of this part of his work considerably clearer. Both central convolutions take part, he thinks, in the building up of "sensory stimuli" flowing to them from the periphery.

Stereognosis is the subject of special consideration, and he believes it is a "gnostic" act; its loss is an "agnosia" depending on many special sensory impulses. These only can be said to be susceptible of anatomical localization in the cortex.

He then considers the cortical affections of vision, the question of visual agnosia and mind-blindness, and denies that any of these conditions are permanent phenomena invariably caused by one localizable anatomical defect, or that they are due to removal of a "centre for visual images."

The seventh chapter is devoted to apraxia, and here von Monakow is diametrically opposed to Liepmann. He believes that apraxia is a complicated group of symptoms and possesses no anatomical localization beyond that of its components. Unilateral apraxia is always associated with gross changes such as paresis or disturbance of "deep sensibility"; bilateral apraxia is accompanied by agnosia or aphasia. Next, he deals with aphasia in the same way. Here he is profoundly influenced by Semon's theory of memory, and the idea that the underlying physical basis of memory depends on "engrams." The most valuable part of this chapter is an analysis of the anatomical conditions found in all the published cases which came to autopsy; these are arranged in groups, accompanied by admirable diagrams. The book closes with a short account of the problem of the localization of mental activities in the frontal lobes.

However much he may disagree with von Monakow's views on cerebral localization, no one, in future, can consider the subject without careful study of the enormous material contained in this book. The diagrams and illustrations are numerous and beautifully executed.

Localisations Cérébelleuses. Par ANDRÉ THOMAS et A. DURUPT. Pp. 197, avec 92 Figures dans le Texte. Paris: Vigot Frères, 1914.

These authors have long occupied themselves with the functions of the cerebellum, and have made several communications on their researches to the Société de Neurologie. The present work is a complete account of their experiments, and the views they now hold on the localization of function in the cerebellum. Any loss of function due to local lesions has the following characters: (1) Active movement is disturbed, impulsion is excessive and too rapid, cessation too slow (dysmetria); (2) it is possible to change the movement of the affected limbs passively, and to place them in various positions without the animal attempting to resist or readjust them; (3) excessive reaction when the limbs are forced into certain positions opposed to those assumed passively; (4) the absence of reaction which should normally occur to certain excitations arriving from the joints.

In how far can it be said that local lesions produce these changes in any one or other part of the body? The authors conclude that distinct centres exist in the cerebellar hemisphere for the upper and lower limbs (for details consult p. 142). Each of these centres can be divided into secondary ones corresponding to a segment of a limb or one articulation. Each presides over movement in a certain direction, and is "hyposthenic" for the muscles which carry it out, but "hypersthenic" for their antagonists. These centres can be called into action by conditions which alter the posture of the limbs, and are responsible for the maintenance of static tone. Any loss of function in one of these centres disturbs the equilibrium of the limb, and thus diminishes the accuracy of movement. Hence dysmetria, adiadococinesia, and other motor troubles found in disease of the cerebellum. Conscious sensations are never affected; the change is a want of reaction to unconscious articular and static afferent impulses. The cerebral cortex remains dominant and tends to supply, to a greater or less extent, the function diminished by the lesion of the cerebellum; but although recovery may appear to be complete, fear or anything which distracts attention will bring out the previous loss of function in the affected limb.

The Brain in Health and Disease. By JOSEPH SHAW BOLTON, M.D., D.Sc., Medical Director West Riding Asylum, &c. Pp. 479, with 99 Illustrations. London: Arnold, 1914.

Dr. Bolton was one of the first to describe the cyto-architecture of the cerebral cortex, and his work on the visual area is well known. The first part of this book, devoted to the minute structure and development of certain

parts of the cortex, contains the fruit of a large amount of individual research. This is followed by an attempt to determine the localization of function in anatomical structures of the cortex; "visuo-sensory," "visuo-psychic," and "psycho-motor" zones are described, and the prefrontal area is held to be the "voluntary-cerebral-associative region." A chapter is devoted to language and thought, and another to feeling, emotion and sentiment; these contain less personal observation.

The second half of the book is devoted to the author's views on cerebral function in mental disease, and the abnormal appearances of the cortex found in the insane. He considers that the brains of some patients show obvious want of development in certain layers, whilst in other cases the deficiency in depth was due to disease. All mental disease is therefore divisible into two groups, "amentia," and "dementia," according to whether the fault is due to want of development or disease. This leads to a somewhat unusual classification of mental disease, of which many examples are analysed from the author's standpoint. The illustrations are extremely good.

An Introduction to the Study of Colour Vision. By J. HERBERT PARSONS, D.Sc., F.R.C.S. Pp. 308, with 75 Figures. Cambridge: University Press, 1915.

At a time when there is much loose writing on the subject of theories of colour vision, this book will come as a valuable reminder of the difficulty of the problem and the great labour expended on attempts at its solution by many devoted workers. Dr. Parsons has attempted to indicate what we know of colour vision from its physical, physiological, and psychological aspects. He shows a profound knowledge of the literature of the subject, and the references he gives to the source of every statement made would alone make his book a valuable addition. After a short introduction he describes the spectrum as seen first by the light-adapted eye, and then by the dark-adapted eye: this opens up the question of luminosity and colour mixture. The fields of vision for colour are given, and he then passes to a consideration of simultaneous and successive contrasts with the phenomena of flicker and fatigue.

Part II is devoted to the facts of colour blindness, and Part III to the chief theories of colour vision. It is impossible for anyone to approach this subject without some predilection to some one or other aspect of the controversy. But, whatever views the author holds, he has succeeded in stating the main theories, and the results of the work based upon them, in a singularly clear light. From his account and from the references he gives, anyone is placed in the position to follow up any of the principal hypotheses. Hering said, "Sterile theories easily relinquish immortality. Fruitful theories hand down their immortal part to their children, whilst their ephemeral shell falls to pieces." Dr. Parsons has indicated the observations upon which each of the great theories has been built up, and shown where knowledge has advanced though hypotheses fail.

Feeble-mindedness, its Causes and Consequences. By HENRY HERBERT GODDARD, Ph.D. Pp. 599. Illustrated by a large number of photographs. New York: Macmillan, 1914.

The author of this book is the Director of the Research Laboratory of the Training School at Vineland, New Jersey, and in this capacity he applied the Binet tests to a large number of feeble-minded girls and boys. In each case an attempt was made to obtain as complete a pedigree as possible of each patient with the help of trained workers, and the larger part of the book is made up of these pedigrees with a short description of the patient's mental and physical condition, illustrated in most cases by photographs. In exactly half the author's cases the feeble-mindedness was hereditary, and in half these patients both parents were feeble-minded. Alcoholic excess, syphilis, illegitimacy and all forms of mental defect were present in enormous excess in these families, and the author is almost inclined to look upon feeble-mindedness as a "unit" character from the hereditary point of view. By no means all cases of mental defect arise from a defective stock: some seem to be sports in a normal family, whilst a larger proportion of the non-hereditary cases are the direct consequence of some infantile disease such as cerebro-spinal meningitis. In the introductory and concluding chapters the conclusions, though dogmatically expressed, are of great general interest, and give a fair idea of the social problems opened up by such work.

Mentally Defective Children. By ALFRED BINET and TH. SIMON. Authorized Translation by W. B. DRUMMOND, M.B., C.M., F.R.C.P., with an Appendix containing the Binet-Simon tests of intelligence, by MARGARET DRUMMOND, M.A. Pp. 180. London: Arnold, 1914.

This is a translation of Binet and Simon's account of how they came to institute their tests for defective intelligence. In places it deals almost entirely with French conditions, and vivacious accounts are given of the difficulties which met them in their campaign for reform. The most useful portion of the book at the present time is the complete list of the tests and a guide to the method of carrying them out.

Syphilis and the Nervous System. By Dr. MAX NONNE. Authorized Translation from the second revised and enlarged German Edition by CHARLES R. BALL, M.D. Pp. 406, with 98 illustrations in the text. Philadelphia and London: Lippincott. No date.

This is a somewhat abbreviated translation of the second German Edition of Nonne's well-known book: it contains all the excellent photographs and diagrams of the original, and strictly follows its readable arrangement. Many of the brilliant little clinical descriptions of cases have been omitted, but the others have been incorporated in the text.

The Intervertebral Foramen, an Atlas and Histologic Description of an Intervertebral Foramen and its Adjacent Parts. By HAROLD SWANBERG. Pp. 101, illustrated by 16 full-page plates. Chicago: Scientific Publishing Company. No date.

The purpose of this little book is to present an accurate description of the right first dorsal intervertebral foramen and its contents in the cat. One specimen only was examined, and the foramen and its contents were cut into sixty sections; but as the nervous structures were not stained differentially no new facts appear from this curious piece of work.

Mental Diseases: A Text-book of Psychiatry for Medical Students and Practitioners. By R. H. COLE, M.D., M.R.C.P. Pp. 353, with 52 illustrations and plates. London: Hodder and Stoughton, 1913.

This text-book will be useful to the student who wants an introduction to the study of insanity. It follows clinical lines, and deals shortly with all the common forms he is likely to meet. The photographs of patients are unusually good, and the final chapters on prognosis and the law of lunacy and mental deficiency are the best in the book.

Orthopædics in Medical Practice. By Professor ADOLF LORENZ and Dr. ALFRED SAXL. Authorized Translation from the German by L. C. PEEL RITCHIE, M.D., F.R.C.S., Ch.M. Pp. 288, with 39 illustrations. London: Bale, Sons and Danielsson, 1913.

This is a good translation carried out by a surgeon who was a voluntary assistant in Professor Lorenz's clinic. The great mass of the book deals with the rectification of deformities and disabilities due to disease of the nervous system; yet its feeblest side is the neurological. The time has past when any surgeon who is not fully imbued with the ideas of modern neurology can hope to write successfully on such a subject.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

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